Active Isolated Stretching: An Investigation of the Mechanical Mechanisms

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ABSTRACT

The Active Isolated Stretching (AIS) technique proposes that by contracting a muscle (agonist) the opposite muscle (antagonist) will relax through reciprocal inhibition and lengthen without increasing muscle tension (Mattes, 2000). The clinical effectiveness of AIS has been reported but its mechanism of action has not been investigated at the tissue level. Proposed mechanisms for increased range of motion (ROM) include mechanical or neural changes, or an increased stretch tolerance. The purpose of the study was to investigate changes in mechanical properties, i.e. stiffness, of skeletal muscle in response to acute and long-term AIS stretching for the hamstring muscle group.

Recreationally active university-aged students (female n=8, male n=2) classified as having tight hamstrings, by a knee extension test, volunteered for the study. All stretch procedures were performed on the right leg, with the left leg serving as a control. Each subject was assessed twice: at an initial session and after completing a 6-week AIS hamstring stretch training program. For both test sessions active knee extension (ROM) to a position of "light irritation", passive resisted torque and stiffness were determined before and after completion of the AIS technique (2x10 reps). Data were collected using a Biodex System 3 Pro (Biodex Medical Systems, NY, USA) isokinetic dynamometer. Surface electromyography (EMG) was used to monitor vastus lateralis (VL) and hamstring muscle activity during the stretching movements. Between test sessions, 2x10 reps of the AIS bent knee hamstring stretch were performed daily for 6-weeks.

Subjects extended the knee significantly further (session 1: 158.4°±12.6; session 2: 173.3°±11.5) after completing the long-term stretching program ($p \leq 0.05$). After a single bout of AIS there was a trend toward increased ROM within the first session, however it was impossible to determine conclusively whether this change was statistically significant, due to changes in the control leg ROM. No significant change was found in stiffness values. In both test sessions hamstring activity was significantly less than VL activity during AIS, when expressed as %MVC. Long-term AIS appears to be effective at increasing ROM. A trend for the immediate benefits is also evident. AIS does not appear to affect mechanical mechanisms because there was no change in stiffness values. The contribution of neural mechanisms is also apparent and requires further investigation.

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CHAPTER 1

INTRODUCTION

1.1 Background

Therapists and athletes regularly include stretching skeletal muscles as part of their rehabilitation and training programs. Stretching is a general term used to describe any manoeuvre designed to lengthen shortened soft tissue structures. Therefore stretching increases the range of motion (ROM), the degree of movement, at the joint associated with the skeletal muscle. Investigations of static, dynamic, ballistic and proprioceptive neuromuscular facilitation (PNF) stretching techniques have identified mechanical and neural mechanisms which may explain the increased ROM. A change in stretch tolerance is a third proposed mechanism to account for the change in ROM due to stretching (Etnyre & Abraham, 1986b; Taylor, Dalton, Seaber & Garrett, 1990; Halbertsma & Goeken, 1994).

Static stretching is a process of slowly lengthening a muscle and then holding it at a constant length for 30-60 seconds. This stretch technique utilizes the mechanical properties of stress relaxation and creep, as well as the neural mechanism of autogenic inhibition to increase ROM (Holcomb, 2000).

Dynamic stretching mimics sport specific movements by using quick controlled movements that are within a normal ROM. Bouncing at the end ROM is avoided. The movement patterns are specific to the sport and are often included as part of the active warm-up because they help prepare the athlete for

athletic competition. The neural mechanism of reciprocal inhibition is utilized to achieve the increase in ROM achieved from dynamic stretching (Holcomb, 2000).

Ballistic stretching involves fast active contractions of the agonist muscle to elongate the antagonistic muscle. The end position is not held which creates a bouncing type of movement that causes the joint to move into an extreme ROM. Ballistic stretching triggers the myotatic stretch reflex, which limits the ability of the antagonist muscle to achieve optimal lengthening (Holcomb, 2000).

Proprioceptive Neuromuscular Facilitation (PNF) stretching uses a combination of active and passive movements with isometric, concentric and eccentric muscle contractions to facilitate reciprocal and autogenic inhibition. These neural mechanisms optimize muscular relaxation, which allows the musculoskeletal structures to achieve a greater ROM (Holcomb, 2000).

These stretch techniques have been thoroughly investigated and have been shown to be effective at improving joint ROM. Another stretch technique, active isolated stretching (AIS), has not been investigated as rigorously. The clinical effectiveness of AIS has been reported (Leimohn, Mazis & Zhang, 1999; Marino, Ramsey, Otto & Wygand, 2001; Middag & Harmer, 2002) but, to my knowledge, its mechanism of action has not been investigated at the tissue level.

1.2 Active Isolated Stretching (AIS)

The basic principle of AIS, as explained by its creator Aaron Mattes, is that by contracting a muscle (agonist) the opposite muscle (antagonist) will relax through reciprocal inhibition and lengthen without increasing muscle tension. AIS

uses multiple repetitions of stretches lasting less than two seconds, allowing the muscle to optimally lengthen without triggering the protective myotatic reflex, which inhibits the stretch potential. It is suggested that a greater stretch is obtained via AIS because of the relaxed state of the antagonist muscle (Mattes, 2000).

The Mattes method is based on the practical application of Wolff's and Sherrington's Laws. Wolff's Law states, "The form of the bone being given, the bone elements place or displace themselves in the direction of the functional pressure and increase or decrease their mass to reflect the amount of functional pressure" (Mattes, 2000). The sheets of fascia that surround muscle have been laid down in a very precise way along the lines of stress within the body and therefore they need to be stretched along the same lines. The positioning of the muscle and tendon is critical in stretching as proper alignment minimizes the tension and friction allowing full elongation to occur.

When a muscle on one side of a joint is contracted, the muscle on the opposite side sends a neurological signal to relax or release. This is Sherrington's Law of reciprocal inhibition and muscle contraction (Mattes, 2000). Inhibition of the reflexive myotatic stretch is also incorporated in the AIS technique with slow, controlled, rhythmic stretches of less than two seconds. Rapid stretching would trigger the myotatic stretch reflex, causing a contraction of the antagonist muscle which would limit the stretch potential.

1.3 Purpose

Current research on the technique of active isolated stretching has focused on the effect of AIS on ROM and comparison with the other stretching techniques. The purpose of the study was to investigate mechanical properties of the hamstring muscle group in response to the AIS technique. The study assessed the acute and long term effectiveness of AIS and observed the underlying mechanism of action.

1.4 Hypotheses

1. Active isolated stretching of the hamstrings will increase ROM at the knee.
2. Mechanical mechanisms will be responsible for the increase in ROM, because the stiffness of the hamstring muscles will be decreased after performing AIS.

CHAPTER 2

LITERATURE REVIEW

In order to investigate the three proposed mechanisms which account for the change in ROM due to stretching, the underlying tissue components must be analyzed. The structure, and the effects of stretching on the structure, of muscle and connective tissue will be addressed. The neural components of muscle and their effect on elongation will also be discussed. Research on the three proposed mechanisms will be presented, as well as the research that has been conducted on AIS and similar stretch techniques.

2.1 Tissue Composition

There is a close association between the contractile elements of muscle, the muscle fibers, and the non-contractile elements of connective tissue, such as fascia. Contractile muscle components are mostly protein based and surrounded by connective tissue at several levels. Connective tissue is made up of various densities and spatial arrangements of collagen fibers embedded in ground substance. Collagen is a fibrous protein that has a very high tensile strength. Collagenous tissue is found in many different structures, including tendons, ligaments, joint capsules, aponeuroses and fascial sheaths, among others (Hunter, 2000).

2.1.1 Muscle Components

The ability of muscle to contract is due to its very organized structure. Actin and myosin myofilaments are responsible for the shortening of muscles. Myofilaments are arranged longitudinally in the sarcomere, which is the smallest contractile unit of skeletal muscles. Sarcomeres are linked successively to form a long, thin strand called a myofibril. A muscle fiber is a cluster of hundreds to thousands of myofibrils that have been grouped together by connective tissue. The myofibrils run the entire length of the muscle fiber and create a cylindrical cell that approximates the diameter of a human hair (Hunter, 2000). Up to 150 muscle fibers are bundled together by connective tissue to create a fasciculus. The muscle belly is composed of numerous fasciculi.

The sarcomere is considered the contractile unit of the muscle. During muscle contraction the length of the sarcomere decreases because the actin filaments slide over the myosin filaments. Conversely, when the muscle relaxes or is passively lengthened, the sarcomere increases in length. The ability of the actual muscle fiber to shorten is due to the series arrangement of the sarcomeres (Hunter, 2000).

2.1.2 Fascia and Tendons

Fascia is a three dimensional fibrous matrix that provides interconnections throughout all cells of the body. There are four levels of fascia that surround muscle: epimysium, perimysium, endomysium and sarcolemma. Epimysium encases the entire muscle belly and is continuous with the tendons at the ends of

the muscle. Perimysium surrounds the bundles of muscle fibers (fasciculi). Endomysium surrounds each individual muscle fiber (Hunter, 2000). The close association of fascia and muscle fibers means the ability of the muscle to elongate is directly influenced by the fascial layers.

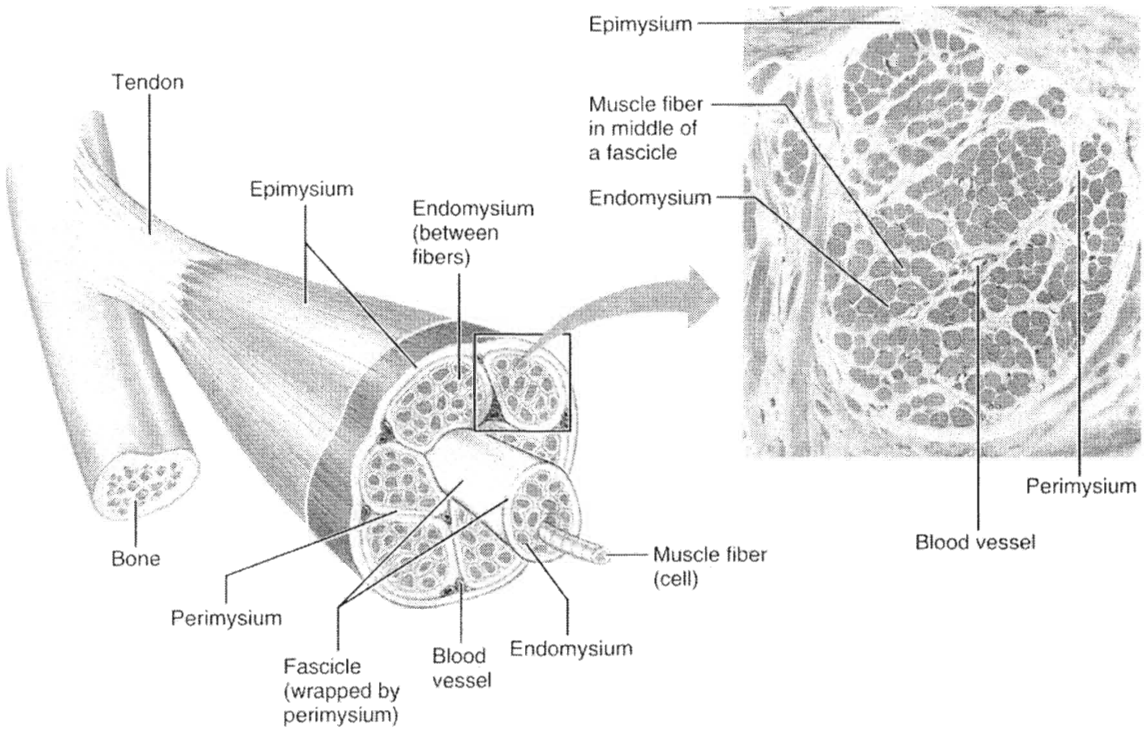

Fig.1 Muscle fiber and fascia layers (Marieb, 2004)

Tendon is a continuation of the endomysium, perimysium and epimysium. Tendon joins the muscle to the periosteum of bone (Figure 1). It is composed mostly of collagen, and a relatively low proportion of elastin. Three protein strands twist around each other in a triple helix to create a procollagen molecule. The helical structure is stabilized by hydrogen bonds between the three strands. The procollagen molecules align end to end, but they do not actually connect. This creates a collagen filament. Filaments align themselves side by side, the

procollagen molecules in one filament overlap the gap between the molecules of the adjacent filament. The parallel arrangement of filaments is called a microfibril. Microfibrils are grouped together to become a fiber and the fibers are bundled together to create the tendon. The strong chemical bonding, called covalent cross-linking, that forms between adjacent collagen molecules throughout the collagen bundle creates the strength of collagen. A complete collagen fiber is not enclosed by a cell membrane, it is just a parallel arrangement of protein strands that have been bunched together in the extracellular space because of a mutual attraction and then chemically cross linked to form a stable structure (Hunter, 2000). Distributed between the collagenous bundles are parallel rows of fibroblasts and a fine reticulum of elastic fibers (LaBan, 1962). Collagen fibers are capable of only a slight degree of extensibility but are very resistant to tensile stress. The small percentage of elastin fibers provides minimal elasticity to the tendon.

When a relaxed muscle is physically stretched most, if not all, of the resistance to stretch is derived from the extensive connective tissue framework and sheathing within and around the muscle, not from the myofibrillar elements (Sapega, Quendenfield, Moyer & Butler, 1981). The parallel arrangement of the fascia layers and muscle fibers allows this to take place. The muscle fiber maintains its normal resting length while the connective tissue reacts to the stretch forces. Change in length when the muscle relaxes or is passively lengthened occurs as the parallel connective tissues slide over the myofibril elements.

2.1.3 Joint Capsule

All synovial joints in the body are surrounded by a joint capsule. The joint capsule is connective tissue composed of collagen and elastin. The capsule joins the articulating bones. Ligaments also surround a joint to provide stability. The structure is similar to that of tendons; they are mostly parallel bundles of collagen with interwoven elastin fibers. However, the higher percentage of elastin fibers and the more random arrangement make ligaments more pliant and flexible (Conroy & Earle, 2000). All joints have a close-packed and an open-packed position. The close-packed position for the joint is when the articular surfaces of the bones are most congruent. To create the firm approximation of the boney structures the joint capsule and ligaments are taut and often twisted. During passive movements these inert structures may provide resistance to limit the end ROM for the joint. The close-packed position for the knee, specifically the tibiofemoral joint, is knee extension with lateral tibial rotation (Hartley, 2000). Therefore, when the knee is fully extended the ligaments and joint capsule are stressed, along with the fascia and tendons of the hamstring muscles. There are no boney limitations to the tibiofemoral motion in the sagittal plane, and as a result, anterior and posterior stability is controlled by the ligaments and muscles around the joint (Hartley, 2000). Therefore, the position of the joint during a stretch maneuver is important as it can determine whether certain structures are affected or unaffected by the stretch.

2.1.4 Relevance to Stretching

Although the amount of joint ROM is determined primarily by the shape and congruency of the articulating surfaces, under normal circumstances connective tissue is the major structure limiting joint motion (Sapega et al., 1981). Relative contributions to the resistance to stretch in the midrange of joint motion are 47% joint capsule, 41% passive motion of muscle, 10% tendon and 2% skin. The restraining effect of the tendons becomes more important towards the extremes of joint motion (Johns & Wright, 1962). The extensibility of tendon and fascia surrounding the muscle belly controls the ability of the muscle-tendon unit to elongate and therefore protects the myofibrils from being stretched to a point of damage (Purslow, 1989). It has been said that tendons act as mechanical buffers to protect muscle fibers from abrupt length changes (McHugh et al., 1999).

There are two different methods by which fascia influences the ability of the musculotendinous unit to elongate. The series elastic component utilizes the longitudinal arrangement of muscle fibers and tendon, the endomysium in particular. The function of the endomysium is to transfer force from the contractile component to the tendon and bone in series (Magnusson et al., 1996c). Connective tissue also surrounds the muscle fibers and has the ability to slide parallel to the muscle fibers creating the parallel elastic component of fascia. The perimysium is suggested to be the principal component in the parallel elastic component. Its role is to distribute stress evenly and to prevent over-stretching (Kubo, Kanehisa & Fukunaga, 2002). When a muscle is passively stretched the

length-tension relationship is used to define passive muscle stiffness (Gajdosik & Bohannon, 1987). It is unclear if the passive muscle stiffness represents the series or the parallel elastic component. A study by McHugh et al. (1999) reports the passive muscle stiffness measurement is a reflection of the extensibility of the tendon-aponeurosis complex, the series elastic component. Conversely, Kubo, Kanehisa and Fukunaga (2001) found that passive stiffness is not related to the extensibility of the tendon structure, but to that of the connective tissue elements in parallel with the muscle fibers. Regardless of which component of the connective tissue it measures, both studies concur that the passive muscle stiffness represents the extensibility of the fascia, not the muscle fiber.

2.2 Mechanical Properties

A change in the mechanical properties of the muscle-tendon complex is one mechanism that has been proposed to explain the increase in ROM that occurs after stretching (Taylor et al., 1990; McHugh, Kremenic, Fox & Gleim, 1998). The mechanical properties characterize how the musculotendinous unit structures respond to a stretch force.

2.2.1 Viscoelasticity

The process of elongating tissue is stretching, while the actual elongation or linear deformation is referred to as stretch. There are two types of stretch: elastic and plastic. Spring-like behavior, where elongation produced by tensile loading is recovered after the load is removed, represents elastic stretch.

Therefore, it is also described as temporary or recoverable (Sapega et al., 1981). Elastic properties are visually represented by Hooke's model of a perfect spring (Figure 2A) (Taylor et al., 1990). Plastic stretch exhibits putty-like behavior. Even after the tensile stress is removed the linear deformation remains. This is described as nonrecoverable or permanent elongation (Sapega et al., 1981). Viscous properties of the tissue permit plastic deformation (Sapega et al., 1981) and are represented by Newton's model of a hydraulic piston known as a dashpot (Figure 2B) (Taylor et al., 1990). Muscle, as with most biological tissue, displays both elastic and plastic, or viscous, properties and therefore is said to behave viscoelastically. Biomechanical models represent viscoelastic characteristics by combining springs and dashpots in series and parallel (Figure 3) (Taylor et al., 1990).

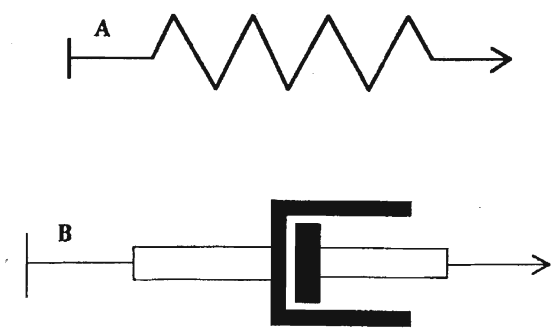

Fig. 2 Visual representation of the elastic (A) and viscous (B) properties of tissue (Taylor et al., 1990)

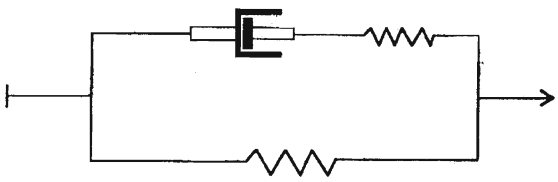

Fig. 3 Visual representation of the viscoelastic properties of tissue (Taylor et al., 1990)

Stretching techniques utilize the characteristic properties of viscoelastic material to achieve elongation of the musculotendinous tissue. Viscoelastic properties include stress relaxation, creep, hysteresis and strain rate dependence. Stress relaxation occurs when the material is stretched and then held at a constant length. The force at that length gradually declines. Creep is characterized by continued deformation at a fixed load. Hysteresis is the variation in the load-deformation relationship that takes place between loading and unloading. The strain rate dependence of viscoelastic material means it exhibits higher tensile stresses at faster strain rates (Taylor et al., 1990).

Stretching techniques should primarily focus on promoting plastic deformation, as the goal is to permanently increase the ROM. The relative proportion of elastic and plastic deformation varies depending on how, and under what conditions, the stretching is performed. Factors that influence the proportion of plastic and elastic stretch are amount of applied force, duration of applied force and tissue temperature. Low-force, long-duration stretching at elevated temperatures favors viscous or plastic deformation. Also, low forces and higher temperatures minimize structural weakening of the tissue (Sapega et al., 1981).

An elevated temperature decreases the stiffness and increases the extensibility of the tissue. It is thought that this mechanism of action in collagenous material occurs due to intermolecular bonds becoming partially destabilized, enhancing the viscous flow properties of the tissue (Sapega et al., 1981). Therefore, to optimize stretching potential, and to prevent damage, a warm-up should be performed before any stretching procedure.

2.2.2 Force-Deformation Relationship

Force and deformation are two critical mechanical properties that are commonly measured when testing tendon structures: an externally applied force, expressed in units of Newtons (N), will cause the structure to elongate (deform), which is expressed in units of meters (m). Hooke's law describes the linear relationship between force and deformation. However, during dynamic loading tendon displays a non-linear viscoelastic response with an initial toe-region followed by a progressively steeper region (Magnusson, Hansen & Kjaer, 2003). A stress-strain or torque-angle curve is often used to visually describe this phenomenon (Figure 4). The stiffness of the material is expressed as the relationship between stress and strain, the slope of the line in the steeper portion of the curve (Magnusson, 1998). As previously discussed, during passive stretching the stiffness refers to the passive extensibility of fascia.

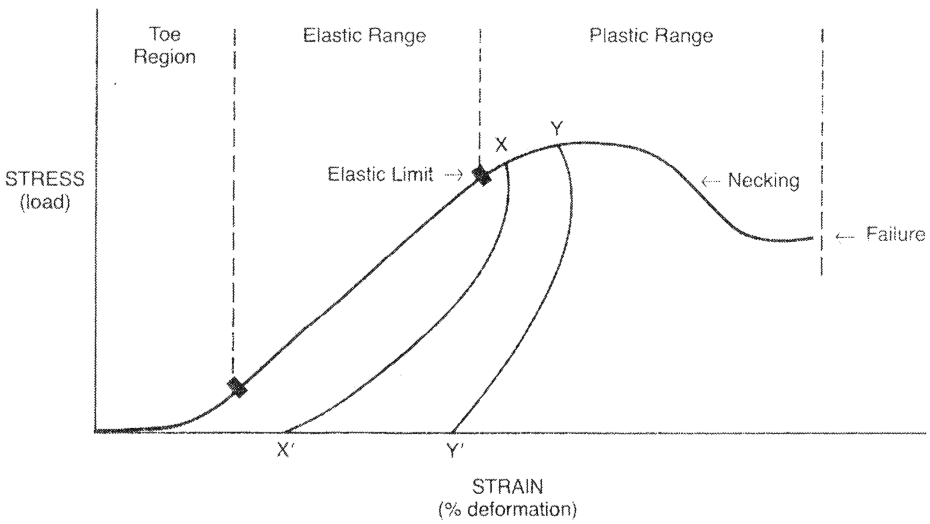

Fig. 4 Stress-strain curve (Kisner & Colby, 1996)

The shape of the curve is due to the underlying composition of the connective tissue. The collagen molecules that comprise connective tissue possess relatively strong intermolecular van der Waals bonds. However, the bonds between the helical procollagen molecules are relatively weak. When a force is applied to these materials, this disproportion in bonding strengths permits adjacent chains of molecules to slip over one another allowing a response to stress. Initially the molecules are disoriented; with application of increasing force the helical composition becomes longitudinally aligned. This change in orientation strengthens the intermolecular bonds and increases resistance to further deformation (LaBan, 1962). Therefore, the molecules become aligned in the toe region and the linear portion represents the actual elongation of the collagen tissue.

2.2.3 Passive Stiffness

In the past, studies have used goniometric ROM measurements to represent flexibility, however this is an invalid method (Gajdosik, 1995). Goniometric results should be reported and interpreted as ROM measurements only, not as measurements of factors that may affect ROM (Gajdosik & Bohannon, 1987). Stress and strain account for the dimensions of the structure, and thereby provide information only about its qualitative properties. Force and deformation provide information about the quantitative mechanical behavior of a structure (Magnusson et al., 2003). However, complex passive movements were found to be difficult to assess reliably. It has been suggested that force

dynamometers should be used to standardize the amount of passive force applied, and thereby decrease the potential for error (Gajdosik & Bohannon, 1987). Nuyens et al. (2000) used repeated passive isokinetic knee movements to investigate the test-retest reliability of torque measurements in healthy subjects. Passive flexion and extension movements were repeated 10 times consecutively with a 5-second rest interval between each movement. The test involved three sets with the angular velocity set at 60, 180 and 300 °/s respectively. After the three sets of 10 consecutive movements were performed the subject was removed from the isokinetic apparatus for 20 minutes, and then repositioned and re-tested. Results demonstrated that torque can be reliably measured during consecutive passive movements. They caution that standardization of patient positioning is essential for the repeatability of measurements on different test occasions (Nuyens et al., 2000).

Resistance to stretch is defined as the passive torque, in Newton meters (Nm), offered by a muscle group during passive elongation using an isokinetic dynamometer (Magnusson, 1998). The isokinetic dynamometer also provides information on the joint angle positioning. These data can then be used to create a torque-angle curve. Stiffness is defined as the change in torque (Nm) divided by the change in position (radians) and is calculated from the slope of the torque-angle curve (Magnusson, 1998). Passive stiffness can be calculated using different mathematical models (Nordez, Cornu & McNair, 2006). A second-order polynomial, a fourth-order polynomial or an exponential model can be used to fit the torque-angle data to calculate stiffness.

The role of gravity during passive movement should be addressed. Gravity opposes knee extension and therefore, unless corrected for the effect of gravity, the torque values for passive resisted extension will be an overestimation. Gravitational moment can be calculated by different methods. The most frequently used technique is an isokinetic assessment, however, a more accurate anthropometric model has been identified (Kellis & Baltzopoulos, 1996). In the isokinetic assessment of gravitational moment the torque resulting from the effect of gravity on the combined weight of the leg and the dynamometer is the gravity effect torque (GET) (Fillyaw, Bevins & Fernandez, 1986). To attain the GET for knee extension the subject is positioned the same as the testing protocol. The dynamometer is set at a speed of 30°/s. The examiner holds the knee in full extension. The subject is instructed to remain completely relaxed and allow the extremity to fall passively against the resistance offered by the dynamometer from full extension through to 90° of flexion. The peak torque ($GET\theta_1$) and the angle (θ_1) of the peak torque during this movement are the important recordings. With these values the GET can be calculated for the angle of knee flexion at which peak torque occurs (θ_2).

$$GET\theta_2 = \frac{GET\theta_1(\cos\theta_2)}{\cos\theta_1}$$

The gravity corrected hamstring peak torque value is calculated by subtracting the $GET\theta_2$ from the uncorrected peak hamstring torque measured by the dynamometer (Fillyaw et al., 1986). This method assumes that gravitational moments measured by the dynamometer are not influenced by the elastic forces developed due to the viscoelastic nature of the connective tissue in the

musculotendinous units of the hamstrings. Furthermore, it is assumed that there is no force exerted by the muscle groups involved because the limb is completely relaxed during the procedure. This, however, is difficult to achieve in a laboratory environment. The gravitational moments obtained by an isokinetic dynamometer have been found to be significantly different from those obtained using anthropometric data because of these assumptions. The anthropometric model is not affected by muscle action factors, and therefore is considered to be a more accurate method for gravitational moment estimation. The anthropometric model uses the following equation to calculate the moment when the leg is in full extension:

$$M = (l \cdot 0.437) \cdot (0.06 \cdot BW)$$

where M = gravitational moment, l = length of the limb (m), 0.437 = COM-proximal joint distance/segment length, 0.06 = leg-foot weight/body weight and BW = body weight (N). The gravitational moment at any other angle A (M_A) can be determined by the following cosine function:

$$M_A = (M \cdot \cos A) \Big/ (\cos 30°)$$

The gravity corrected hamstring torque values can be calculated by subtracting the M_A from the uncorrected hamstring torque values measured by the dynamometer (Kellis & Baltzopoulos, 1996).

The validity for the passive stiffness measurement has been investigated (Kubo et al., 2001; McHugh et al., 1998). Both investigations found a negative correlation between passive stiffness and maximal ROM. Hence, a decrease in passive stiffness was related to an increase in maximal ROM. Therefore, it

seems reasonable to suggest that the passive stiffness measured in the studies can be an index of flexibility (Kubo et al., 2001).

2.2.4 Biomechanical Effects of Stretching

The following studies have confirmed that the increase in ROM after stretching was due to a change in the mechanical properties of the muscle-tendon unit.

The muscle-tendon unit responds viscoelastically to tensile loads and is not influenced by reflex activity (Taylor et al., 1990). Clinically relevant biomechanical stretching properties in an entire muscle-tendon unit under passive stretch were experimentally evaluated. The study of rabbit extensor digitorum longus and tibialis anterior muscle-tendon units was divided into three parts. In the first part characteristics of repeated stretching of muscle-tendon units to a predetermined length were examined. This is similar to a cyclic stretching technique to a given length. The second section involved muscle-tendon units being stretched repeatedly to the same tension and held at a fixed length. This section simulated the technique of static stretching. The final part of the investigation examined how varying the stretch rates and denervating the muscle affects muscle-tendon stretching. Sustained muscle-tendon unit elongation occurred with the application of simulated static and cyclic stretching techniques. The viscoelastic properties of stress relaxation, creep and hysteresis were found to be responsible for the change in tissue length. In other words, a change in the mechanical properties should allow for a greater ROM and greater

flexibility of a joint. The denervated muscles responded similarly to the innervated muscles, therefore the muscle response to stretch can be explained by viscoelastic properties alone, exclusive of stretch reflexes. The stretch reflex may exist during muscle stretching techniques, but in this investigation it had no significant force contributions (Taylor et al., 1990).

It was also noted that for both stretch techniques the greatest changes in the muscle-tendon unit occurred in the first four stretches. The ideal number of repetitions may have been influenced by the magnitude of the stretching force or the duration of the hold. However, a minimal number of stretches appear to be most effective in muscle-tendon unit elongation. Any subsequent stretches will still bring about length increases, but these increases will be small and less significant (Taylor et al., 1990).

McHugh et al.'s (1998) data explains musculoskeletal flexibility in mechanical terms rather than by neural theories. Passive mechanical force and stretch induced contractile responses to stretch were examined to see if they limited maximum straight leg raise (SLR) range of motion. Sixteen recreational athletes performed a SLR stretch with the knee braced in full extension. The end point was when the subject reported discomfort. Throughout the movement a load cell was attached to the ankle to measure torque and surface electrodes recorded the activity of the rectus and biceps femoris muscles. An electrogoniometer reported the hip flexion ROM. Torque-ROM curves were plotted for zero ROM to maximum ROM and back to zero ROM. The results of the study suggest that the SLR test was primarily a measurement of the passive

mechanical forces resisting hip flexion motion. The parallel elastic component accounted for 79% of the variation in hip flexion ROM. A lack of EMG response during the stretch indicates that the perception of discomfort during slow passive stretching precedes any reflex contractile response. It was suggested that reflex activity would contribute to the resistance of the stretch if the ROM had continued beyond the point of discomfort or during a rapid stretch (McHugh et al., 1998).

McHugh et al. (1999) examined the effect of passive muscle stiffness on symptoms of muscle damage after eccentric exercise. The SLR stretch was used to classify subjects as stiff, normal or compliant. The subjects then performed 6 sets of 10 isokinetic submaximal eccentric actions of the hamstring muscle group. Greater symptoms of muscle damage were reported in subjects with stiff hamstring muscles, and attributed to the sarcomere strain theory of muscle damage. They consider the passive muscle stiffness to be a reflection of the tendon-aponeurosis extensibility. The tendon is supposed to act as a mechanical buffer by absorbing lengthening strain to protect the muscle fibers. It is proposed that muscle damage occurs when a rigid tendon-aponeurosis complex transfers the strain imposed by active lengthening to the muscle fibers.

Reid and McNair (2004) were the first to investigate muscle stiffness in the new ROM achieved after a stretch program. Forty-three school-aged students participated in the study, 23 subjects in the intervention group performed a stance phase static hamstring stretch five days per week, once per day, for 3 repetitions of a 30 second hold. Twenty subjects in the control group did not stretch over the 6-week intervention period. The experimental group increased in

knee extension ROM, passive resistive force and stiffness after the six weeks. No significant differences were observed in the control group data for the same variables. The increase in stiffness that accompanied the new ROM, 10° of knee extension, provides evidence that changes in structural characteristics of the tissue had occurred as a result of the stretch program. An increase in muscle fiber length due to the addition of sacromeres in series and an increase in the amount of connective tissue in the muscle are two suggested mechanisms for the increase in stiffness, as they have been documented in studies involving animals (Herbert & Balnave, 1993; Williams & Goldspink, 1973).

2.3 Active Isolated Stretching

The Mattes Method of active isolated stretching has been practiced for the last 30 years. The creator, Aaron Mattes, developed this therapeutic myofascial technique to promote the functional and physiological restoration of muscles, tendons, ligaments and joints to facilitate healthier fascial planes. The technique ostensibly uses active movement and reciprocal inhibition to achieve optimal flexibility. The stretch is most effective when it is performed in 1.5 to 2 seconds; this prevents the reflexive contraction of the antagonist muscle. A specific muscle or group of muscles is identified and precise localized movements are implemented to isolate the stretch of the muscle. The muscle is stretched slightly beyond the point of light irritation by the application of mild pressure, less than 0.5 kg of resistance. The pressure is released and the muscle is returned to the starting position in which the muscle is at resting length. The procedure is

repeated for a prescribed number of repetitions, 5-15, depending on the muscle (Mattes, 2000).

2.3.1 AIS Research

Research specifically involving AIS is limited to three studies, each employing a different protocol. The studies have focused solely on the effect of AIS on ROM and comparison with other stretching techniques. To my knowledge no investigations have focused on the underlying mechanisms of action or the effect at the tissue level.

Leimohn et al. (1999) used active straight leg raise performance to compare the effect of active isolated and static stretch training. Fifteen male and fifteen female subjects, ranging from 18 to 25 years, were tested. Maximum iliofemoral flexion ROM was measured goniometrically with the knee braced in full extension. The AIS and static stretch groups performed nine supervised training sessions over a 3-week period. The AIS group raised the leg to active end ROM and then used a rope to pull it to passive end ROM. The stretch was held for 2 seconds and was repeated 14 times. In the static stretch group the heel was raised off the ground to a height where end ROM was achieved. The static stretch was performed once and held for 30 seconds. Both groups showed significant improvements in ROM. However, the AIS group improved significantly more than the static stretch group. The results indicated that AIS training produced greater ROM gains than static stretching, for these procedures (Leimohn et al., 1999).

Marino et al. (2001) also compared the effects of active isolated and static stretching. Twenty-four female and six male subjects, aged 22.6 ± 1.3 years, were included in the study. Sit and reach and goniometric hip flexion ROM were used to evaluate both techniques, as well as a control group. The stretch intervention groups performed 60 seconds of stretching three times per week, for 13 weeks. Only goniometric measurement of hip flexion for AIS was found to have significant changes. It was suggested that accuracy of the instrument may have effected the change in goniometric measurement but not the sit and reach measurement. It was concluded that a low dose flexibility intervention, 3 sessions per week of only 60 seconds per session, was insufficient to elicit measurable changes in ROM (Marino et al., 2001).

Another comparison of active isolated and static stretching techniques found no difference in their effectiveness (Middag & Harmer, 2002). College-aged recreational athletes actively warmed up for 5 minutes and then performed either the AIS or the static stretching procedure once a day, 5 times per week for 3 weeks. Both groups showed statistically significant increases in hamstring ROM compared to the baseline measurements. However, the groups did not differ greatly in their improvements as the static stretching group increased 8% while the AIS group improved 11%. These results show that minimal time and effort is required to improve hamstring ROM in healthy active individuals. Traditional static stretching was just as effective as the AIS technique (Middag & Harmer, 2002).

2.3.2 PNF Techniques

Due to the limited amount of research on the actual AIS technique it is necessary to review other techniques that closely resemble the AIS procedures. In the late 1940s PNF stretching was developed by Herman Kabat, MD, based on Sherrington's concepts of muscle facilitation and inhibition. The techniques were originally used to treat neuromuscular disorders by restoring strength. More recently PNF methods have been developed to increase ROM. The most frequently used techniques are hold-relax (HR), contract-relax (CR) and agonist contract-relax (Houglum, 2001). HR uses an isometric contraction of the muscle to be stretched followed by lengthening. CR uses a concentric contraction of the muscle to be stretched followed by lengthening. The agonist contract-relax method, is also referred to as Contract-Relax Agonist-Contract (CRAC). CRAC uses a concentric contraction of the opposing muscle followed by passive lengthening of the muscle to be stretched, and is very similar to AIS. The contraction of an agonist muscle to initiate reciprocal inhibition in the antagonist muscle is the basis for both techniques. The techniques vary in the timing and application of external force to promote lengthening. There have been several studies investigating PNF methodology and effectiveness.

Etnyre and Lee (1987) published a review summarizing the findings of previous flexibility studies. Based upon practical applications and experimental procedures among various comparative investigations, PNF methods were found to be more effective and efficient than static or ballistic stretching techniques in producing greater ROM. The CRAC technique was found to be the most effective

PNF method to increase the ROM. They also conclude that future studies should continue for an extended period with regular measurements to determine if and when differences between methods occur (Etnyre & Lee, 1987).

Holt, Travis and Okita (1970) compared the effects of ballistic, static and CRAC stretching techniques on hamstring flexibility via a sit and reach instrument. The CRAC technique positioned the subject lying supine with both knees extended throughout the procedure. The subject flexed the hip to raise the leg until a stretch was felt in the hamstrings. An examiner stabilized the leg while the subject performed a 6-second isometric contraction of the hip extensors. This was immediately followed by a contraction of the hip flexors to increase the elongation of the hip extensors, the hamstrings. Three contractions were repeated and followed by a 10-second rest period, after which the other leg was stretched. This process of alternating between legs was continued for two minutes. A second CRAC stretching technique was also performed from a standing position. The same procedures were followed with the subject flexing the trunk forward to initiate a stretch of the trunk extensors. The trunk extensors contracted isometrically and then the trunk flexors concentrically contracted to increase the ROM in the trunk extensors. The CRAC method produced significantly greater flexibility scores than the ballistic and static stretching techniques (Holt et al., 1970).

The effect of static, CR and CRAC stretch techniques on hip ROM was investigated in men, aged 17-26 years (Cornelius & Hinson, 1980), and in females, aged 16-23 years (Moore & Hutton, 1980). The CRAC technique

involved the subjects starting in supine lying position with the leg held straight. In the study by Moore and Hutton (1980) a brace was applied to passively hold the knee in extension. The subject actively flexed the hip to raise the leg from the ground, a 3-6 second isometric contraction of the hip extensors was followed by a concentric contraction of the hip flexors resulted in further elongation of the hamstrings. A greater degree of hip flexion was found using the CRAC stretch technique compared to the static and CR methods (Cornelius & Hinson, 1980; Moore & Hutton, 1980).

The CRAC stretching technique was also found to be the most effective in increasing ROM in the soleus muscle. Static, CR, and CRAC techniques were performed on separate days by 12 subjects. Both PNF techniques were more effective that static stretching and the CRAC technique was found to be superior to the CR method of stretching (Etnyre & Abraham, 1986a).

2.4 Neural Pathways

The effect of muscle stretching in humans has been discussed as a function of the passive mechanical properties of the muscle-tendon unit. However, it has also been suggested that the increase in joint ROM is due to neural components that are activated by some stretch techniques (McHugh et al., 1998).

The central nervous system (CNS), the brain and the spinal cord, collects information from afferent nerves and initiates the body's response via efferent nerves. Proprioception is the body's ability to consciously and unconsciously

respond to the afferent stimuli regarding position (Houglum, 2001). Located within joints, muscles and tendons, are specialized sensory receptors called proprioceptors. They are sensitive to pressure and tension, and relay information concerning muscle dynamics to the CNS. Two significant mechanoreceptors are the muscle spindles and Golgi tendon organs (GTOs) (Harris & Dudley, 2000). These mechanoreceptors are responsible for the activation of neural reflexes that influence the ability of the muscle-tendon unit to elongate during a stretch technique.

2.4.1 Muscle Spindles

Muscle spindles are modified fibers, called intrafusal fibers, which run parallel to the normal, extrafusal, muscle fibers. They provide information concerning muscle length and the rate of change in length. When a muscle is stretched, deformation of the muscle spindle occurs and activates the sensory neuron. An impulse is sent to the spinal cord where it synapses with a motor neuron innervating the same muscle, and a sensory neuron innervating the reciprocal muscle. Thus, muscle spindles control the myotatic stretch reflex and reciprocal inhibition (Harris & Dudley, 2000).

2.4.2 Golgi Tendon Organs (GTO)

GTOs are located near the myotendinous junction in tendons. They are attached in series, end to end, with extrafusal muscle fibers. When the musculotendinous unit is stretched, tension in the muscle and tendon increases

causing the discharge from the GTO to increase. The sensory neuron of the GTO activates an inhibitory interneuron in the spinal cord, which in turn synapses with, and inhibits, a motor neuron serving the same muscle. The result is a reduction in tension within the muscle and tendon. It is a protective mechanism against the development of excessive tension, called autogenic inhibition (Harris & Dudley, 2000).

2.4.3 Stretch Reflexes

Activation of the proprioceptors initiates a neural response which may be advantageous or disadvantageous to tissue elongation. The myotatic stretch reflex occurs when rapid stretching of a muscle lengthens both the extrafusal muscle fibers and the muscle spindles. This deformation activates the sensory neuron of the spindle, which sends an impulse to the spinal cord. In the spinal cord, the sensory neuron synapses with motor neurons. This results in an activation of the motor neurons that innervate the same muscle, and causes the muscle to contract. The resulting contraction is roughly equal in force and distance to the original stretch stimulus. This protective reflex prevents injury that could result from a rapid increase in muscle length. The myotatic stretch reflex inhibits a muscle's ability to lengthen and therefore a stretching technique should avoid activating this reflex (Holcomb, 2000).

When a muscle contracts the increase in muscular tension is sensed by the GTO. The sensory neuron of the GTO sends a signal to the spinal cord, where it synapses with a motor neuron which inhibits the contracting muscle. The

GTO causes the muscle to reflectively relax, this is called autogenic inhibition. Tension built up during an active contraction stimulates the GTO, causing a reflexive relaxation of the muscle, which optimizes any subsequent passive stretching (Holcomb, 2000).

Another reflex that helps a stretching technique to optimally lengthen tissue is reciprocal inhibition. When the sensory neuron from the muscle spindle synapses in the spinal cord with a motor neuron it also synapses with another sensory neuron which innervates the reciprocal muscle. Activation of the sensory neuron causes inhibition in the reciprocal muscle. Therefore, contraction of the agonist muscle causes a reflex relaxation of the antagonist muscle. The principle of reciprocal inhibition is also referred to as Sherington's Law (Holcomb, 2000).

2.4.4 Neural Impact of Stretching

Reciprocal inhibition is the key concept in both AIS and the CRAC technique of PNF stretching. Investigations of reciprocal inhibition have found mixed results, and its role in contributing to musculoskeletal flexibility remains controversial. The following are studies which support the idea that reciprocal inhibition promotes muscle relaxation and therefore allows for greater flexibility.

Etnyre and Abraham (1986b) investigated the neuromuscular influences of different stretching procedures. The neural mechanisms considered in the rationale of stretching methods involve the activation of muscle spindles and GTOs. The level of inhibitory or excitatory influence from these proprioceptors on the motor pool excitability may be observed by evaluating the Hoffman reflex (H-

reflex). The H-reflex is an electrical stimulation of a nerve that recreates the myotatic stretch reflex that occurs when a muscle is stretched. The only difference is that the H-reflex bypasses the muscle spindle (Palmieri, Ingersoll & Hoffman, 2004). Each of 12 subjects was randomly assigned to a treatment-order group. The techniques of static, CR and CRAC stretching were performed on separate days for each subject. A marked and lasting suppression of the motor pool excitability, in agreement with the studies of reciprocal inhibition, was demonstrated by the CRAC-PNF stretching technique. It is believed that optimal muscle elongation occurs when the muscle is in a relaxed state, thus when greater motor pool inhibition is evident. The results of this investigation suggest that PNF methods, particularly those involving reciprocal activation of muscles, provide the greatest potential for muscle lengthening (Etnyre & Abraham, 1986b).

The rationale that contraction of the muscle being stretched is inhibited by antagonist contraction is supported by Etnyre and Abraham (1988), and is consistent with those studies which indicate that the CRAC technique shows greater ROM gains. They used fine wire and surface electrodes to investigate the muscle activity in plantar flexor and dorsiflexor muscles during a stretch technique similar to the CRAC method to stretch the plantar flexors. Examination of recordings from the wire electrodes showed no activity in the soleus muscle during tibialis anterior contraction. This indicated that reciprocal inhibition occurred during the CRAC technique. EMG recorded with surface electrodes contained inter-muscle cross-talk and therefore appeared to show suppressed

reciprocal inhibition phenomena. The researchers suggested that care must be taken when using surface electrodes (Etnyre & Abraham, 1988).

Surface electrodes are often used because they are less invasive, easy to apply and more accessible to researchers. A disadvantage is that they are said to only be effective for superficial muscles and cannot detect signals from small muscles. However, they are very effective for clinical assessments of time and magnitude of activation and studies of general gross relaxation to tenseness (Basmajian & Deluca, 1985).

The motorneuron pool excitability during stretching of muscle by contracting the antagonistic muscles was evaluated by Guissard, Duchateau and Hainaut (1988). Static stretching and CR techniques for the triceps surae muscles were performed by 28 healthy physical education students. The tendon (T-) and H-reflexes were analyzed during the stretching procedure. The T- reflex occurs when an external force is used to increase tension in a tendon, thus activating the neural pathways that are normally stimulated by the GTO during a stretch procedure. Inhibition of the motor neuron pool excitability was present during all three stretch techniques. Reciprocal inhibition was the rationale used to explain the result; contraction of the opposing muscle inhibited the muscle that was targeted to be stretched. It was noted that the inhibition of the motor neuron pool stopped as soon as the stretching technique ceased (Guissard et al., 1988).

In 2001 Guissard, Duchateau and Hainaut further investigated the concept of reciprocal inhibition during stretch techniques. They concluded that both pre- and postsynaptic mechanisms caused reduced motoneurone excitation during

stretching. EMG was used to record activity in the soleus muscle in response to electrical stimulation of the tibial nerve at the popliteal fossa (the H- reflex), and at the ankle (the exteroceptive reflex). The results suggested that during small-amplitude stretching the decreased H-reflex should be related to presynaptic mechanisms and thus located at pre-motoneural level. It was proposed that the postsynaptic inhibitory mechanisms contributed to the decreases in both reflexes and the motor-evoked potential (MEP), for large-amplitude stretching. Inhibitory afferents from the GTO are thought to play a greater role in decreasing motoneurone excitability via Ib fibers during large-amplitude stretching. This is consistent with the fact that the GTOs, which respond mainly to the muscle fiber contraction force, are hardly sensitive to the mechanical tension of passive stretching and only seem to be activated during large amplitude stretching (Guissard et al., 2001).

Shindo, Harayama, Kondo, Yanagisawa and Tanaka (1984) also investigated the Ia inhibition mechanism by analyzing the suppression of the soleus H-reflex by stimulation of the peroneal nerve. During dorsiflexion, Ia inhibition of the soleus motoneurones was demonstrated in four out of the five subjects. The mechanism for inhibition of the pathway during plantar flexion was considered to be inhibition of the Ia interneurone of the flexor side by the Ia interneurone of the antagonist extensors (Shindo et al., 1984).

Investigations have also found results that disagree with the concept of reciprocal inhibition. In 1980, Moore and Hutton used EMG to investigate the relative level of muscle relaxation achieved during the application of static and

modified PNF stretch procedures. Participants were female gymnasts, 17-23 years of age. It was thought that gymnasts would understand and perform the stretch training techniques with less apprehension because of their daily experience with flexibility training. The subjects performed static, CR and CRAC PNF stretching procedures while one knee was braced in full extension and their ankle was attached by a cable to a pulley system. Hip ROM was assessed by a goniometer, electrogoniometer and potentiometer, an indirect measurement via the pulley system. EMG data were simultaneously collected for the rectus femoris and semitendinosus muscles. The CRAC method produced the greatest increase in ROM and was found to elicit the greatest muscle activity, compared to the other two techniques. Thus, full muscle relaxation was not imperative for effective stretching. This suggested that the notion of reciprocal inhibition causing increased stretch potential should be discarded (Moore & Hutton, 1980).

Static stretching, hold-relax (HR), agonist contract (AC) and hold-relax-agonist contract (HRAC) stretching techniques were investigated in plantar flexors by Condon and Hutton (1987). The subjects were seated while their foot was secured to a footplate and torque device for producing dorsiflexion of the ankle. ROM was measure by an electrogoniometer and EMG activity was recorded for the soleus and tibialis anterior muscles. The investigation found similar increases in ROM following the performance of each of the stretching procedures. The techniques involving agonist contractions, AC and HRAC, had smaller H-reflex amplitudes, suggesting possible reciprocal inhibition during the agonist contraction. If reciprocal inhibition had occurred, the effects were masked

by other neuronal input, as shown by the existence of high tonic EMG activity levels. The findings are in agreement with the findings of Moore and Hutton (1980). An agonist contraction assisting the stretch significantly increased EMG activity and, therefore, muscle relaxation during stretch appears to have no effect on ROM achieved (Condon & Hutton, 1987).

Hamstring activity and knee extension ROM was used to evaluate three modified PNF stretch techniques in the study by Osternig, Robertson, Troxel and Hansen (1987). All three techniques were performed by the subjects, 6 men and 4 women, aged 23-36, after adequate warm-up was completed. Surface electrodes recorded EMG activity of the vastus lateralis and biceps femoris while the stretch-relax (SR), CR and ACR techniques were performed. The subjects were in the supine position with the left thigh strapped in extension to stabilize the pelvis and the right thigh strapped in full hip flexion. The ROM of the right knee was recorded when the knee was extended to the point of restriction. The CR and ACR techniques did result in sufficient relaxation of the hamstrings, the muscles opposing knee extension, to overcome facilitation generated by stretch. During active knee extension the hamstrings exhibited 55% of the EMG activity produced by a maximal voluntary contraction (MVC). However, knee ROM was greatest under these conditions. These results suggest that reciprocal inhibition does not occur with these PNF techniques, and muscle relaxation is not required to attain maximal ROM (Osternig et al., 1987). It was noted that when a task requires precision or when subjects are untrained in the task, strong agonist excitation produces simultaneous activity in the antagonist (Person, 1958).

Similarly, when subjects felt more confident and in control of the procedure they were more willing to extend their knee further in spite of antagonist resistance and expressed discomfort (Moore & Hutton, 1980).

In 1990 Osternig, Robertson, Troxel and Hansen used the identical testing procedures to test the effects of the three PNF techniques in different athlete populations. They found no difference in the effect on hamstring muscle activation and knee extension ROM in endurance athletes versus high intensity athletes versus a control group. The ACR procedure produced the most EMG activity and 9-13% more knee joint ROM than the other two procedures. It was concluded that decreases in muscle activity are not strongly related to increases in joint ROM. Also, 64-84% of total ROM increases occurred during the first of the two phases for all of the PNF techniques. Therefore, ROM gains may be limited during a given stretch session regardless of repetitions (Osternig et al., 1990).

During slow (30°/s) isokinetic knee extension involving maximal volitional quadriceps contraction Åagaard et al. (2000) found considerable antagonist co-activation of the hamstring muscles. Knee joint moments for 16 sedentary males were collected using a Kin-Com dynamometer. The subjects were seated to perform two types of isokinetic knee extension tests. In the first test the quadriceps actively generated an extension moment and in the second test the hamstrings eccentrically contracted to generate a flexion moment. EMG signals for the vastus lateralis, vastus medialis, rectus femoris, biceps femoris and semitendinosus were collected. The data suggested that during isokinetic knee

extension, co-activation of the flexors of the knee occurs due to a neural pathway. It was suggested that this hamstring co-activation is to assist the mechanical and neurosensory functions of the anterior cruciate ligament. Near full knee extension the contractile forces of the quadriceps induce anterior tibial shear and excessive internal tibial rotation. The antagonist hamstring moments potentially counteract these movements (Åagaard et al., 2000).

2.4.5 Fast Limb Movements

Results from Marsden, Obeso and Rothwell (1983) support the opinion that the action of the antagonist muscle is to provide a counter-acting braking force, to assist in halting a fast movement. Viscoelastic components of a joint are sufficient to stop movement, below a certain threshold speed. However, at the extremes of rotation viscoelastic properties offer greater resistance to movement, therefore, larger movements that approach end ROM require smaller antagonist activity. Thumb flexion was studied by analyzing the EMG activity of the agonist flexor pollicis longus and antagonist extensor pollisis longus muscles. Elbow extension was also assessed by examining the agonist triceps and antagonist biceps EMG activity. The following variables were measured from each record: the amplitude and peak velocity of movement, the time of onset and cessation of the agonist and antagonist EMG bursts, and the integrated EMG activity during the agonist and antagonist bursts. For quick movement for a short distance the antagonist burst was large and followed quickly after the agonist activity, and when the movement was slower, over a longer distance, the antagonist burst

was small and late. Antagonist activity does not occur if it is not necessary, as when the subject knows there is a definite end-stop (Marsden et al., 1983).

Wierzbicka, Wiegner and Shahani (1986) also investigated the role of agonist and antagonist muscles in fast arm movements. The characteristic pattern of EMG activity of the agonist and antagonist muscles during fast movement was originally described as a "triphasic pattern". The agonist muscle provides the initial burst of activity which is followed by a silent period during which the antagonist becomes active. After the antagonist ceases the agonist is activated again. The first agonist burst provides the force to accelerate the limb. The function of the antagonist is generally thought to provide the braking force decelerating the limb. The function of the second agonist burst has not been studied in detail, but it has been suggested that it secures the movement at the target position. This study found that by activating both agonist and antagonist muscles the same peak displacement which was attained by activating only one muscle can be obtained more quickly. They concluded that a primary function of the antagonist is to control movement time. In order to produce fast movements, a large agonist torque followed by an equally large or larger antagonist torque is required. It has been shown that eccentric contractions produce more force than similarly-activated concentric contractions. Thus, EMG activity of an agonist and antagonist could be the same, but because the antagonist is eccentrically contracting its torque will be greater than the agonist torque. The amount of the second agonist burst is proportional to the difference between the first agonist contraction and the antagonist contraction. Therefore, the third burst will increase

as the imbalance between the agonist and the antagonist torques increases (Wierzbicka et al., 1986).

2.4.6 Mechanical and Neural Factors

Studies by Taylor et al. (1990) and McHugh et al. (1998) concluded that only mechanical factors affected musculotendinous elongation, and that the neural factors were irrelevant. A few studies have reported the increase in ROM due to stretching as a combination of both the mechanical and neural components of the muscle-tendon unit.

In 2003 Magnusson et al. reported on the role of tendons with respect to the neuromuscular control of limb positioning. Joint position is partially controlled by the afferent feedback from muscle spindles, which lie in parallel with muscle fibers. Muscle spindle activation is proportional to the change in length of the muscle fibers. The myotatic stretch reflex that inhibits muscle elongation occurs when muscle spindles are triggered. The muscle fibers are in series with tendons, therefore tendon compliancy will affect the activation of muscle spindles. In a very stiff or short tendon the length change of the whole muscle-tendon complex will be sensed by the muscle spindle. Minimal changes in length and a small change in the ROM of the joint will activate the muscle spindle. Alternatively, if a muscle has a very long and extensible tendon then the tendon may lengthen considerably before the muscle fibers are affected. As a result, more elongation, and a greater ROM, will occur before the muscle spindle is

activated (Magnusson et al., 2003). Therefore, both mechanical and neural mechanisms affect the muscle-tendon unit extensibility due to stretching.

A critical review of the literature by Shrier (2004) investigated the hypothesis that stretching improves performance. MEDLINE and Sport Discus search engines were used to find studies related to stretching and performance. Twenty-three articles investigating the acute effects of stretching were analyzed. These studies indicate that the viscoelastic behaviour, i.e. stiffness, of the musculotendinous unit was decreased when assessed within one hour of the stretching protocol. It was also noted that in most studies EMG was affected, suggesting that a neurologic mechanism may also be present, however further investigations are required (Shrier, 2004).

Guissard & Duchateau (2006) also consider the response to stretching to be attributable to both mechanical and neural factors. In contrast to strength training, in which neural adaptations are followed by muscle hypertrophy, they believe that PNF stretch training involves mechanical adaptations followed by neural adaptations.

2.5 Pain Tolerance

The third proposed mechanism to account for the increase in ROM due to stretching is a change in pain tolerance. The underlying mechanism of action of an altered pain tolerance has not been confirmed. It has been hypothesized that the muscle, and therefore the subject, is able to tolerate a greater exerted moment, and as a consequence, more elongation before a sensation of pain or

stretch is felt (Halbertsma, van Bolhuis and Goeken, 1996). There are conflicting results regarding the relevance of pain tolerance to stretching.

2.5.1 Pain Tolerance is Irrelevant

The stretch tolerance of muscles could not explain the increase in hamstring passive ROM that was observed by Krabat, Laskowski, Smith, Stuart and Wong (2001). They consider the contributing factors to muscle flexibility to be a combination of mechanical and neural components. The popliteal angle ROM on the non-injured knee of 15 subjects undergoing arthroscopic knee surgery was assessed preoperatively, intraoperatively under anesthesia and postoperatively after recovery from anesthesia. The subjects were in a supine position with the injured hip and knee in full extension. For the starting position the non-injured hip and knee were flexed to 90°, and the end point popliteal angle was measured when the knee was extended until a firm endpoint was obtained. A hand dynamometer was used to ensure that equal pressure was being applied at each firm endpoint. The mean popliteal angle in the intraoperative period was found to be significantly greater than the mean popliteal angle in the postoperative period. Also, the group of subjects who received spinal anesthesia showed a greater increase in the mean intraoperative popliteal angle than the subjects who received epidural or general anesthesia. The results may be explained by the fact that spinal anesthetics directly interact with nerves at the spinal cord level providing the densest neural blockage. The afferent signals that were produced during hamstring stretching were unable to synapse with the

efferent nerves. An increase in popliteal ROM was measured because the deregulation of the efferent signals to the hamstring muscles resulted in relaxation, allowing more elongation. The data appear to demonstrate a neurological contribution to hamstring length which is independent of pain (Krabat et al., 2001).

2.5.2 Pain Tolerance is Relevant

The effect of a 4-week CR stretch training program was tested with an instrumented SLR set-up, by Halberstma and Goeken (1994). They tested the extensibility, stiffness and EMG activity of the hamstring muscles before and after the training program. Extensibility was defined as the ability of a muscle to allow elongation, more specifically the ROM over which the limb could be passively moved. Passive stiffness was defined as the ratio of the change in passive muscle moment to the change in muscle stretch (Δmoment/Δangle). Fourteen subjects, classified via the toe-touch test as having short hamstrings, were divided into a stretching group and a control group. The stretching group performed the CR procedure for the hamstrings twice daily for 10 minutes, with one session at 0900 hours and the other session at 2000 hours. Both pre- and post-measurements for each individual were made at the same time of day, as extensibility may be altered through the day (Halberstma & Goeken, 1994). The data obtained showed a slight but significant increase in the extensibility of the hamstrings, as well as a significant increase in the stretching moment tolerated by the passive hamstring muscles. However, the passive stiffness remained the

same. It was concluded that the stretching program did not lengthen or decrease stiffness of short hamstrings. The only change was an increase in the stretch tolerance of the subjects (Halbertsma & Goeken, 1994).

In 1996 Halbertsma et al. carried out a similar study. The 10 subjects in the stretching group performed one, 10-minute static stretching intervention. The hamstrings were stretched statically, from a standing position one leg was raised on a table with the knee extended while the subjects flexed at the trunk, for 30 seconds. The subject then relaxed, the leg still extended resting on the table with the body upright, for 30 seconds. This was repeated for the duration of 10 minutes. The results showed no significant change in the passive muscle stiffness curve with respect to the pre-stretch stiffness curve. Once again they attributed the increase in extensibility and maximum muscle moment to an increase in stretch tolerance. In their opinion, the muscle tolerated the greater moment and as a consequence more elongation was obtained after the stretching procedure (Halbertsma et al., 1996).

Magnusson et al. (1996a) examined EMG activity, passive torque and stretch perception during static and CR stretch techniques. Passive torque (Nm), measured by a dynamometer, was used to quantify the resistance to stretch offered by the hamstring muscle group during passive knee extension. Ten male recreational athletes, aged 29.4 ± 4.1 years, were seated with their trunk perpendicular to the seat and the thigh resting on a specifically constructed pad so that it was elevated 30° - 45° from horizontal. The lever arm of the dynamometer was attached slightly proximal to the lateral malleolus. The

positioning ensured that none of the subjects were able to reach full knee extension during the stretch maneuver, so as to apply tension only to the muscle-tendon unit without involvement of the posterior joint capsule. Gross electrical activity of the hamstring muscles was recorded by the surface electrodes placed mid-way between the gluteal fold and the knee joint. The knee was passively extended to an angle which provoked a sensation of tightness in the posterior thigh. This was used as the final position for the stretch maneuvers. For the dynamic testing, two protocols were followed for both the static and CR stretches. The constant angle protocol passively extended the knee to an angle 10° below the final position where it was held for 10 seconds before continuing to extend to the final position, where it was held for 80 seconds. In the variable angle protocol the knee was passively extended and held for 10 seconds at 10° below the final position. The knee was then further extended until the subject experienced pain in the posterior thigh and pressed a safety switch that instantaneously stopped the lever arm. For the static stretch technique the subjects remained relaxed throughout the protocol. For the CR technique the subjects performed a 6-second isometric hamstring contraction while the leg was held 10° below the final position, during both testing protocols. The data showed that for the constant angle protocol the response of the hamstring muscle was similar with respect to passive torque and low level EMG activity for both the CR and static stretching. For the variable angle protocol, the CR stretching procedure maximal tolerated joint angle and passive torque were found to be greater than the static stretching values. The low level EMG activity remained

unchanged. The data suggested that the viscoelastic and EMG response was unaffected by the type of stretch maneuver, although it was thought that PNF stretching altered stretch perception (Magnusson et al, 1996a).

Another study by Magnusson, Simonsen, Åagaard, Sorensen and Kjaer (1996b) concluded that the increase in ROM achieved from a 3-week static stretch training program was due to an increased stretch tolerance. There was no change in the mechanical or viscoelastic properties of muscle. This study involved seven female subjects, aged 26.0 ± 6.0 years, who only occasionally participated in recreational sports. The measuring techniques and patient positioning were identical to the previous study (Magnusson et al., 1996b), however the stretch protocols varied slightly. For each subject only one leg was tested, while the other served as a control. The final position for this study was determined on the first day and was used for the pre- and post-training stretch protocols. In the first protocol the knee was passively extended to the final position and held for 90 seconds. Protocol two required the patients to close their eyes. The dynamometer passively extended the knee until the patient indicated the onset of pain, at which point they pressed a switch instantaneously stopping the lever arm. Throughout both testing protocols the subjects were instructed to remain completely relaxed. The training program involved two sessions per day, and the stretch was held for 45 seconds with a 15-30 second rest period between five repetitions. The stretch required the subject to sit with the control knee flexed and hip slightly abducted to ensure hamstring relaxation. The stretch leg was extended in front of the subject with the hip in a neutral rotation. The stretch was

initiated with the subject leaning forward with a straight back until a 'stretch' sensation was experienced in the posterior thigh. The results of the study showed the stiffness remained unchanged. The maximum joint ROM and corresponding passive torque increased, and it was concluded that this was most likely due to an increased stretch tolerance (Magnusson et al., 1996b).

Magnusson, Simonsen, Åagaard, Boesen, Johannsen and Kjaer (1997) used the same protocols as described above to examine the hamstring muscles in endurance-trained athletes with varying flexibility. Eighteen male elite-level orienteers were classified as tight, n=10, or normal, n=8, based on a clinical toe-touch test. Data from protocol one showed that the tight subjects' final angle and stiffness was lower than the normal subjects'. During the hold phase of protocol one the tight subjects had a lower peak and final torque than the normal subjects, but the torque decline was similar. Protocol two results indicated that the tight subjects reached a lower maximal angle, torque and stiffness than the normal subjects. In the common range, the stiffness was greater in the tight subjects. It was concluded that individuals with a restricted joint ROM on a toe-touch test, had stiffer hamstrings and a lower stretch tolerance. Tight and normal subjects showed similar viscoelastic stress relaxation at the point of maximal stretch sensation, indicating that all subjects could benefit from a single static stretch (Magnusson et al., 1997).

Once again, the role of stretch tolerance in limiting stretch was examined by Magnusson, Åagaard, Simonsen and Bojsen-Moller (2000). The same protocols as previously discussed were carried out using seven flexible and six

inflexible orienteers as subjects. The results indicated that the flexible subjects attained a greater angle of stretch with an accompanying greater tensile stress than the inflexible subjects, due to an apparent greater tolerance of the externally applied load (Magnusson et al., 2000).

In addition to investigating the acute effects of a stretching bout, the critical review by Shrier (2004) examined the long term effects of regular stretching. The nine studies that were analyzed concluded that three to four weeks of stretching only affected stretch tolerance.

Investigations of static, dynamic, ballistic and PNF stretching techniques have identified three mechanisms to account for the change in ROM due to stretching; mechanical, neural and a change in stretch tolerance. The AIS technique has not been investigated as rigorously, and current research has focused on the effect of AIS on ROM and comparison with the other stretching techniques. This study investigates tissue properties of human skeletal muscle in response to the AIS technique for the hamstring muscle group.

CHAPTER 3

METHODOLOGY

3.1 Subjects

Participants (female n=8, male n=2) were recreationally active university aged volunteers. To participate in the study they had to classify as having tight hamstrings via the standing toe touch (Kippers & Parker, 1987) and knee extension with hip flexed (Bandy & Irion, 1994) tests (described below). They were also free of any lower back, hip or knee pathology, by self-report. Participants were allowed to continue with their normal sports activities, however were not allowed to take part in any other form of formal flexibility training, such as yoga or Pilates. The potential risks and benefits of participation in the study were explained. They were informed of the procedures to be used as well as the purpose of the study and that they could withdraw at any time without penalty. Written informed consent was obtained from the participants before they began any testing and the study had received clearance from the Brock University Ethics Board.

3.2 Testing Procedures Summary

The subjects were required to complete two test sessions: an initial testing session which investigated the acute affects of AIS and a session following the completion of a 6-week AIS program which provided information on the long term effects. An outline of testing procedures can be seen in Figure 5.

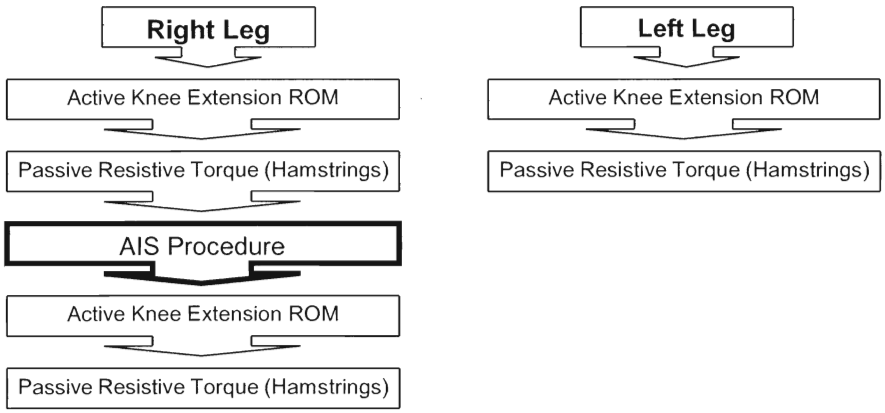

Fig. 5 Summary of the testing procedure for each test session.

Potential participants who contacted the researcher by phone or email after reading a poster announcing the study, were instructed to complete a self screening standing toe touch test to determine if they met the requirements of the study. In the standing toe touch test the participant bent forward from a standing position while holding their knees in extension. Volunteers whose finger to ground distance was greater than 0 cm classified as having tight hamstrings (Halbertsma & Goeken, 1994; Magnusson et at., 1997) and they were invited to participate in the study.

The first testing session began with an assessment of the hamstring flexibility of the participant via a repetition of the standing toe touch test and the completion of a knee extension with hip flexion test. Finger to ground distance of a standing toe touch was measured and recorded for each subject. The knee extension with hip flexion test required the participant to be positioned supine with the right hip and knee supported in 90° of flexion. With the hip held at 90° of flexion the examiner passively extended the tibia to the terminal position, which is

the point at which the subject reported a feeling of discomfort or tightness in the hamstring muscles or when the examiner perceived resistance to stretch. A goniometric measurement was used to determine the amount of knee extension. Full hamstring flexibility was considered to be 0° of knee extension. Having greater than 30° loss of knee extension operationally defined the subject as having tight hamstrings (Bandy & Irion, 1994). Subjects must have classified as having tight hamstrings in both tests to continue in the study. Body weight (kg) was measured on a scale and leg length (m) (lateral femoral epicondyle to malleolus) was measured using a standard metric tape and recorded.

The Delsys Bagnoli-4 EMG system (Delsys Inc., Boston, MA) was used to monitor the electrical activity of the vastus lateralis (VL) and hamstring muscle group. The skin was prepared by shaving, lightly abrading and cleaning with alcohol. Gel was applied between the skin and the electrode to ensure low impedance (Osternig et al., 1990). Electrodes were placed mid-way between the gluteal fold and the knee crease to monitor the hamstrings. An electrode was placed on the muscle belly of the VL muscle, which is approximately two-thirds of the way down on a line from the anterior superior iliac spine to the superior lateral patella (Seniam Project Management Group, n.d.). Muscle activation was recorded throughout the AIS procedure. Following the AIS sets a maximal voluntary contraction (MVC) of the quadriceps and hamstrings was collected. The knee was fixed at 90° of flexion for a 5-second contraction. The muscle activity between second 1 and second 3 of the 5-second contractions was

averaged for three trials and reported as the MVC. All EMG values are reported as the root mean square (RMS).

After the application of the electrodes the Biodex System 3 Pro (Biodex Medical Systems, Shirley, NY) isokinetic dynamometer back rest and knee attachment were set up for the participant. The back rest was set at 90° for all the subjects, however at that setting the subject's hip flexion angle was greater than 90°. Additional support in the form of a pillow placed between the subject's back and the chair back was required to ensure 90° of hip flexion was maintained throughout the procedure (Figure 6). The height and depth of the back rest were adjusted to align the axis of the dynamometer with the estimated knee joint centre of rotation. The knee flexion-extension attachment was positioned proximal to the malleoli and the lower leg was secured to the dynamometer with straps. Chest straps stabilized the upper body and the lap strap stabilized the pelvis. The thigh strap was not fastened, to allow participants who could reach full knee extension to flex the hip towards the chest to adequately stretch the hamstrings. The chair and leg attachment locations were recorded to ensure the same set up was followed for the second test session.

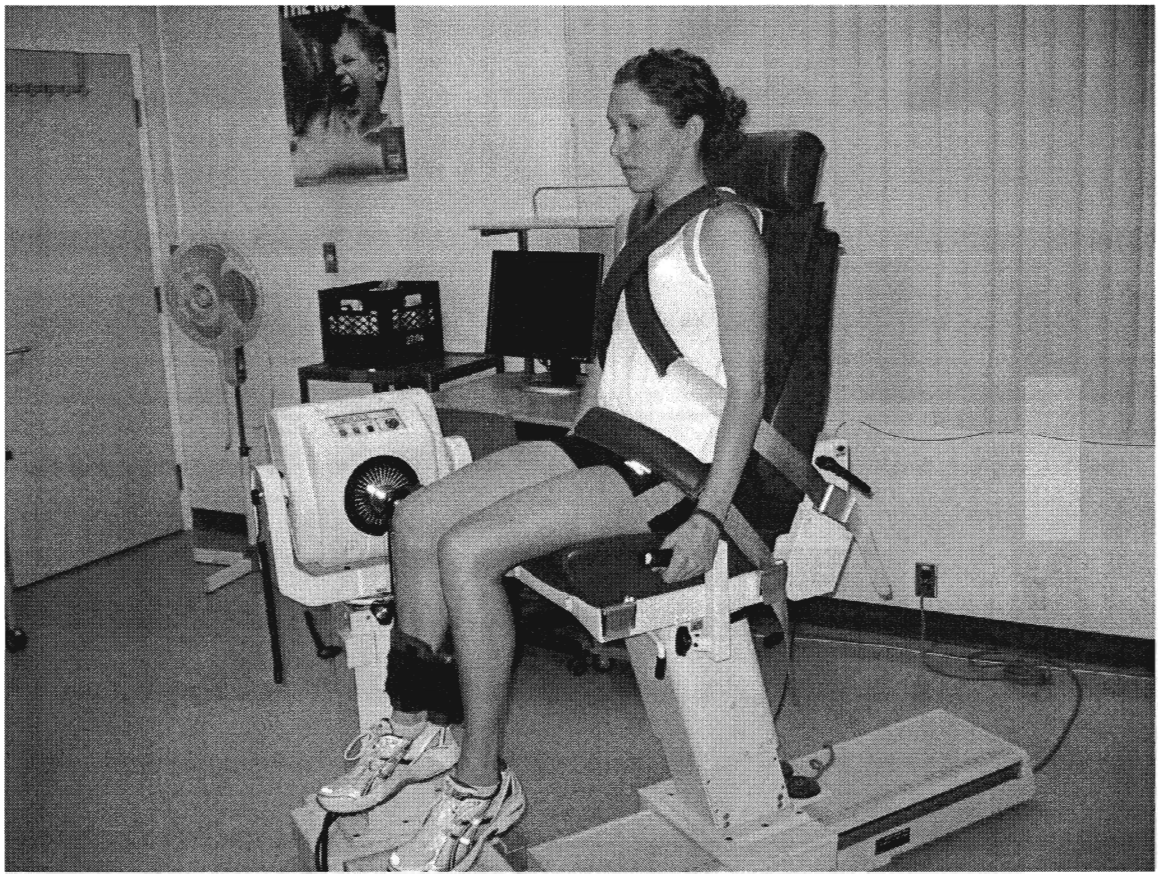

Fig. 6 Biodex chair set-up

After the Biodex was prepared, all participants completed a warm up on a stationary bike for 5 minutes, at a moderate resistance and speed. The initial baseline values for knee extension ROM and passive resistance to stretch were determined. The AIS technique was then recreated with the subject seated on the Biodex. ROM and passive resisted torque were reassessed after the stretch technique was performed.

After completing the 6 week AIS training the second testing session was performed following the same procedures as the first session. For each subject only the right leg underwent the AIS intervention program. The left leg served as

a control. ROM and passive resisted torque of the left leg were assessed in both test sessions, but the AIS program was not performed.

3.3 Range of Motion

For both test sessions knee extension end ROM values were recorded before and after the stretch procedure, to determine if AIS had an effect on ROM. While positioned on the Biodex the participant was instructed to actively extend the knee. The point at which they felt a light irritation in the posterior thigh was considered the end ROM. An 11-point Likert scale (0-10) was used to classify the stretch sensation. A 10 was a painful sensation while 0 was completely relaxed. The subject was instructed to stretch until only light irritation was felt in the hamstrings, which corresponded to 8 out of 10 on the scale. ROM of the right leg was measured twice in each test session, before and immediately after performing the AIS procedure, for a total of four ROM values. The ROM of the left leg was measured once each session.

3.4 Passive Resisted Torque

Passive resisted torque measurements for the right leg were performed before and after the AIS procedure in each test session, and once each session for the left leg. The isokinetic dynamometer was used to assess the passive resisted torque (Nm) of the hamstring muscles during knee extension. The passive mode of the Biodex was programmed to extend the knee at a speed of 5°/s from the starting position in approximately 90° of flexion, to the end ROM for

each subject, which had been previously recorded. The data collected from this procedure were gravity corrected (Kellis & Baltzopoulos, 1996) and then used to create a torque-angle curve from which the stiffness of the hamstring muscles was determined. Stiffness was calculated for the final 10% of the movement which represents the new, stretch induced ROM. A change in stiffness in the new ROM would indicate that changes in the structural characteristics of the tissue had occurred (Reid & McNair, 2004).

3.5 Active Isolated Stretching

The AIS technique immediately followed the initial ROM and passive resisted torque measurements. The end ROM was set to the maximum available ROM of the dynamometer. The starting position was approximately 90° of knee flexion. The isokinetic mode was programmed to allow the knee to flex and extend at a speed of 210°/s. This speed removes resistance of the dynamometer to movement and allows the stretch procedure to be completed in the required amount of time. A metronome set at 90 beats/min provided the subject with a rhythmic beat to follow, and ensured that the entire duration of each stretch was less than 2 seconds. The testing procedure mimicked the bent knee hamstring AIS stretch technique (Mattes, 2000). The participant actively contracted the quadriceps to extend the knee to the point of light irritation of the hamstrings, 8 out of 10 on the stretch sensation scale. The examiner provided the overpressure force by manually assisting the knee extension end ROM, which also ensured that the knee extended to a point of stretch. If the participant was able to reach

full knee extension without irritation they were allowed to bring the hip closer to the chest, while maintaining the fully extended knee, until the stretch sensation was felt. The participant then flexed the knee to return to the starting position. Each participant performed 2 sets of 10 repetitions with a 30 second rest period between the sets.

3.6 AIS Intervention Program

Each participant performed a 6 week AIS training program using the bent knee hamstring stretch technique, as outlined in the AIS manual (Figure 7) (Mattes, 2000). Participants were instructed to warm up before performing the stretch technique using their normal sports activities or a stationary bike at a moderate speed and resistance for 5 minutes, if it was available. Otherwise, they were instructed to jog in place or skip for 5 minutes. For the stretch procedure participants assumed a supine position with left knee flexed so the foot was on the ground. The right hip was semi-flexed to a degree which allowed the knee to fully extend. The starting position of the knee was approximately 90° of flexion. The quadriceps of the right leg contracted to fully extend the right knee. The hip remained in the same semi-flexed position unless the knee was completely extended without the stretch sensation in the hamstrings, at which point the hip could be flexed closer to the chest. When the knee had been actively extended to the point at which a light irritation, 8 out of 10 on the stretch scale, was felt in the hamstrings the participant applied a gentle overpressure to the leg via a rope wrapped around the ankle and foot. After application of the overpressure the

participant flexed the knee to the starting position, the hip remained stationary in the semi-flexed position. The entire stretch procedure was performed in less than 2 seconds, and the repetitions were performed rhythmically. Daily, each participant performed 2 sets of 10 repetitions with 30 seconds of rest between the sets, as recommend by Mattes. They were required to complete a checklist to keep track of their progress and to provide the researcher with a log indicating that the stretch training program was successfully completed.

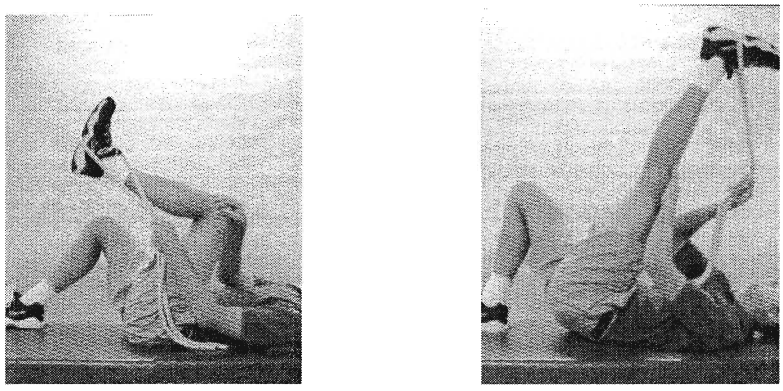

Fig. 7 AIS bent knee technique (Mattes, 2000)

3.7 Gravity Correction

The torque measurements obtained by the isokinetic dynamometer were gravity corrected before creating the torque-angle curve. An anthropometric model was used to calculate the gravitational moment for the lower leg. The following equation was used to calculate the moment when the leg was in full extension:

$$M = (l \cdot 0.437) \cdot (0.06 \cdot BW)$$

where M = gravitational moment, l = length of the limb (m), 0.437 = position of the centre of mass on the long axis of the segment, relative to the knee joint, 0.06 =

weight of the leg-foot segment relative to total body weight and *BW* = body weight (N). The gravitational moment at any other angle *A* (M_A) was determined by the following cosine function:

$$M_A = (M \cdot \cos A) / (\cos 30°)$$

The gravity corrected hamstring torque values were calculated by subtracting M_A from the uncorrected hamstring torque values measured by the dynamometer (Kellis & Baltzopoulos, 1996).

3.8 Data Treatment

Torque and joint ROM was recorded every 10 msec during all procedures on the Biodex. Every tenth data point was used for data analysis. The passive resisted torque values were gravity corrected (Kellis & Baltzopoulos, 1996). A fourth-order polynomial model was used to fit the torque-angle data (Nordez et al., 2006) and stiffness was determined for the final 10% of the ROM (Reid & McNair, 2004) using a custom MatLab program (The MathWorks Inc., Natick, MA).

For the ROM and stiffness values, a 2-way ANOVA (session x time) and Tukey post-hoc tests were used to determine differences between means. A paired t-test was used to analyze the differences between the right (experimental) and left (control) leg values at the beginning of each test session, and to analyze the left leg between test sessions. ANCOVA was used to assess the effect of changes in control leg values on right leg differences produced by the stretching program. An alpha level of 0.05 was considered significant.

Statistica for Windows (Statsoft Inc, Tulsa, OK) software was used for data analysis.

VL and hamstring EMG was collected at 1000 Hz and a bandwidth of 20 to 450 Hz was used. All raw EMG signals were stored on a PC. Muscle activity collected during each set of the AIS procedure was analyzed, for both sessions by determining the root mean square (RMS) values. VL and hamstring activity were expressed as a percentage of the MVC. Differences between the %MVC of the VL and the hamstring muscles within and between test sessions were assessed by an ANOVA and Tukey post-hoc tests were used to determine significant differences. An alpha level of 0.05 was considered significant. Results are reported as mean ± standard deviation.

CHAPTER 4

RESULTS

4.1 Subjects

Ten (female n=8, male n=2) recreationally active volunteers participated in the study. Analysis using ROM data from a pilot study showed that this number of subjects would provide adequate statistical power. All subjects were free from any lower body pathology, and classified as having tight hamstrings according to a knee extension with hip flexion test. Subject characteristics are summarized in Table 1. The knee extension test values are number of degrees short of full knee extension (180°).

Table 1. Subject Characteristics (mean ± standard deviation)

Subjects	Age (yr)	Weight (kg)	Knee Extension Test (°)
n = 10	20.8 ± 1.8	69.4 ± 11.2	40 ± 7

All subjects completed a log book indicating that the stretching intervention program was performed as required, daily for 2 sets of 10 repetitions with 30 seconds of rest between the sets.

4.2 Range of Motion

Figure 8 displays the maximal knee extension position of the subjects' right leg. There was a significant difference between the pre- (158.4° ± 12.6) and post-AIS (167.9° ± 7.1) position within the first test session. There was also a significant difference between the pre-AIS position in the first and second (173.3° ± 11.5) sessions. There was no significant difference between the right and left

leg in either session (Figure 9). This is attributed to an unexpected increase in ROM of the subjects' left leg (control). Figure 9 displays the significant difference for the left leg position between the first (161.1° ± 9.0) and second (168.8° ± 8.0) test session.

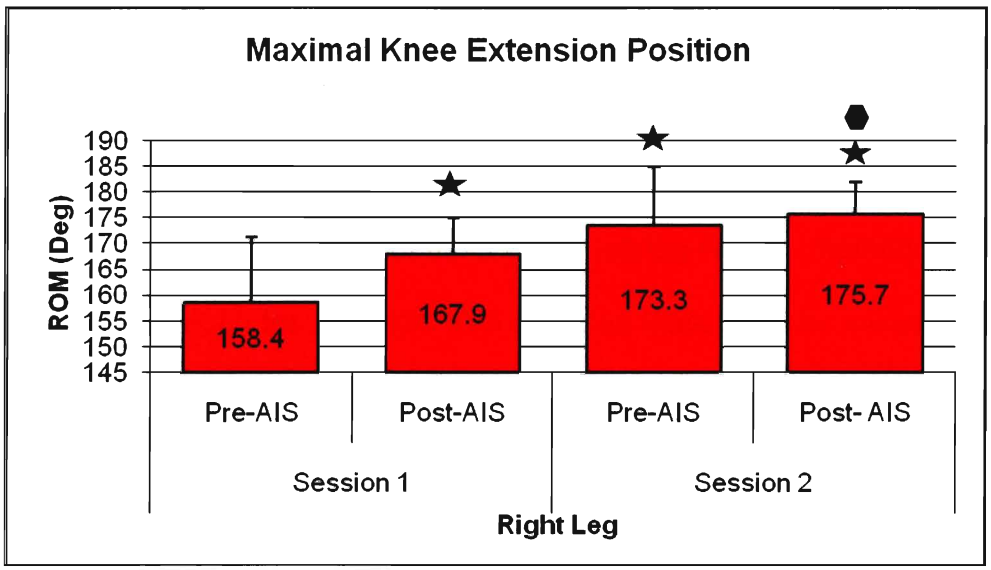

Fig. 8 Maximal knee extension position for the right leg (mean ± SD)
★ *Significantly different from Session 1 Pre-AIS (p< 0.05)*
⬢ *Significantly different from Session 1 Post-AIS (p< 0.05)*

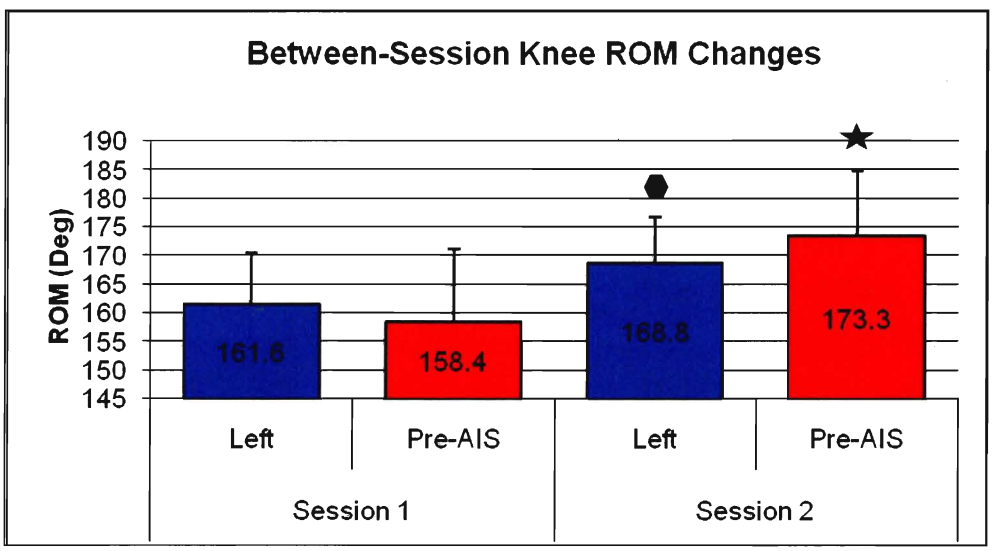

Fig. 9 Left leg and right leg ANCOVA results (mean ± SD)
★ *Significantly different from Session 1 Right leg (p< 0.05)*
⬢ *Significantly different from Session 1 Left leg (p< 0.05)*

The left leg ROM was only measured once per test session. Therefore, the left leg was used as a covariate to assess changes in the right leg ROM values between the test sessions. An ANCOVA revealed there was still a significant difference between the starting right leg ROM (158.4° ± 12.6) in the first session and the starting (173.3° ± 11.5) ROM in the second session (Figure 9).

Consequently, to analyze the right leg ROM within the test sessions with the left leg as a covariate we assumed that the left leg ROM remained unchanged within each test session. The adjusted least squares means for the right leg ROM are shown in Figure 10. The right leg ROM increased, though not significantly, following the first AIS bout (p=0.063), and also following the 6 week training protocol (p=0.052), compared to the pre-AIS (160.7° ± 6.5) position in the first session.

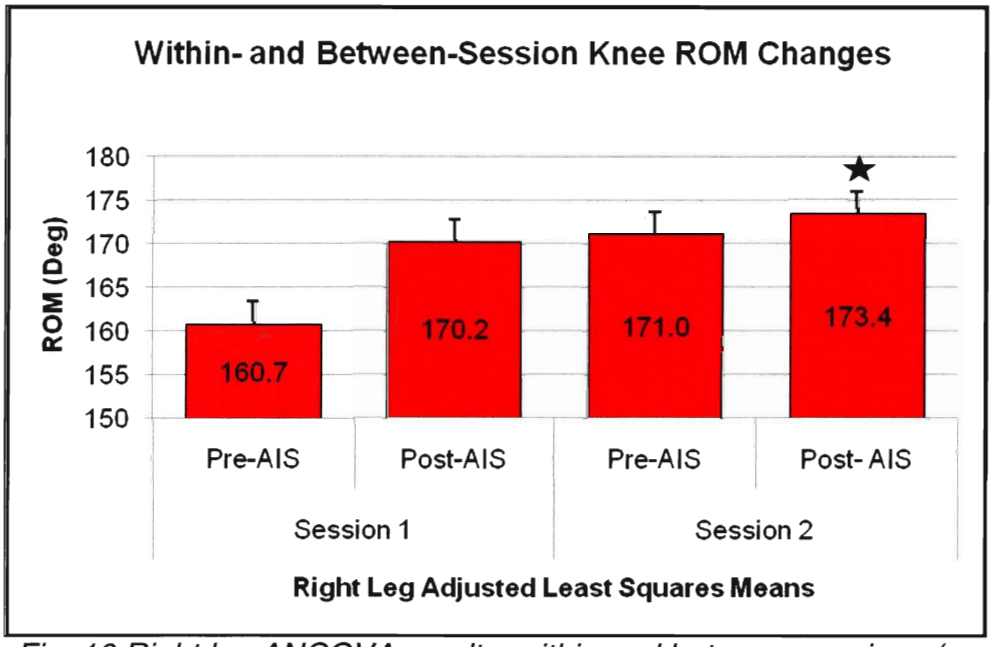

Fig. 10 Right leg ANCOVA results, within and between sessions (mean ± SD)
★ *Significantly different from Session 1 Pre-AIS (p< 0.05)*

4.3 Stiffness

Figure 11 shows the torque-angle curve for a single subject. The blue dots are the raw values from the Biodex. The red line shows the data after gravity correction and the fourth order polynomial fit. Stiffness was calculated over the final 10% of the knee extension ROM. This is depicted by the green line. Figure 12 displays the mean stiffness values (Nm/deg) for the right and left leg of all the subjects. There were no significant differences in stiffness values.

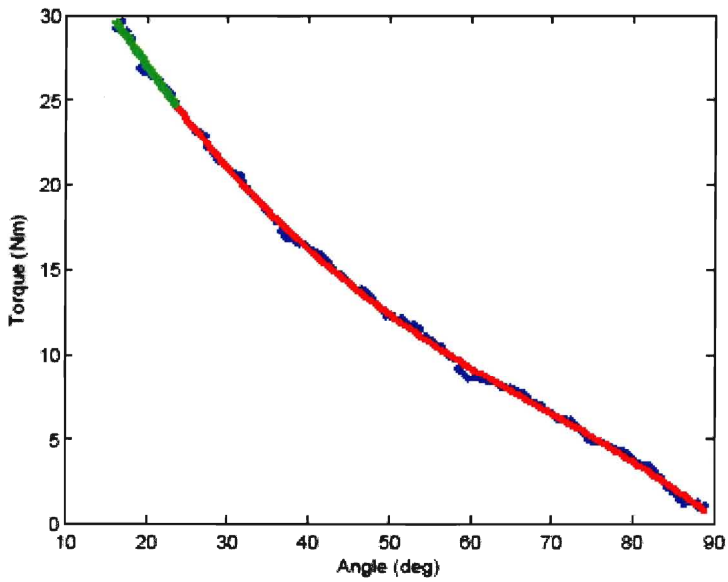

Fig. 11 Torque-angle curve for a single subject.

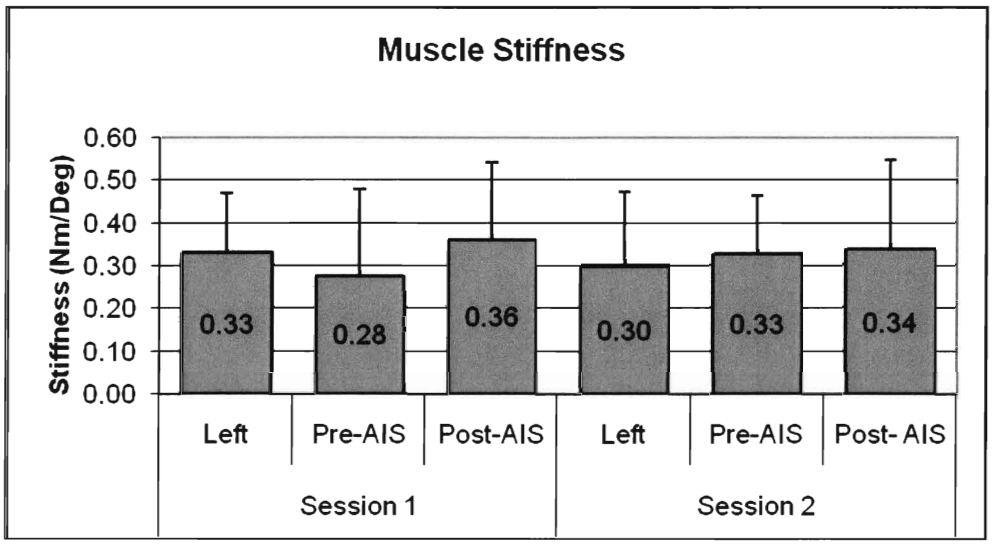

Fig. 12 Stiffness over the final 10% of right knee extension ROM (mean ± SD)

4.4 EMG

Subjects performed 2 sets of 10 repetitions of the AIS bent knee hamstring stretch in each test session. The %MVC of the VL and the hamstring muscles during each set of the AIS procedure is displayed in Figure 13.

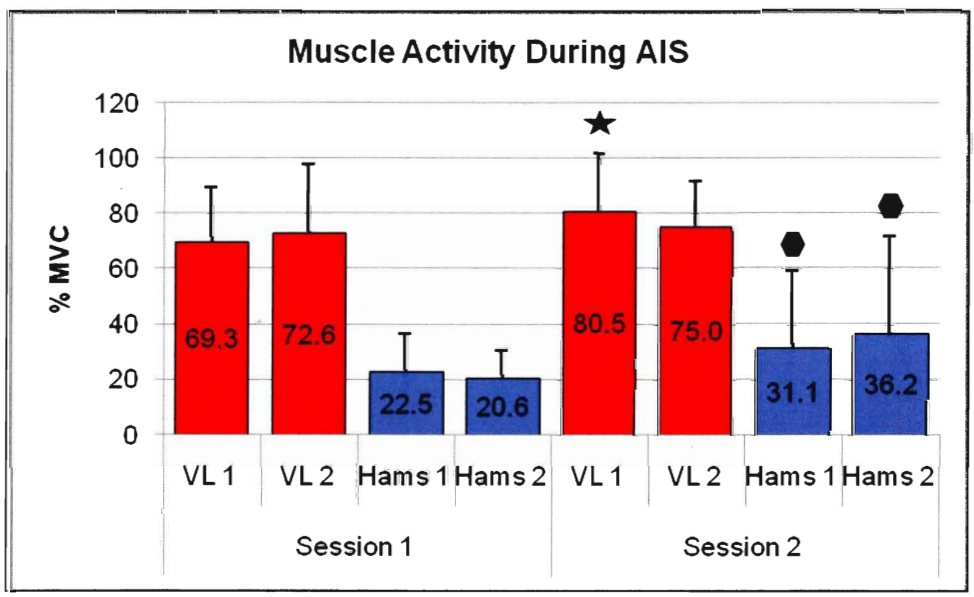

Fig. 13 Muscle activity during the AIS procedure (mean ± SD)
★ *Significantly different from VL1 and VL2 Session 1 (p< 0.05)*
⬢ *Significantly different from Hams 1 and Hams 2 Session 1 (p< 0.05)*

The VL activity in the first set of the second session was significantly greater than the other VL values. The VL was significantly more active than the hamstring group during the performance of AIS, within and between sessions. The hamstring activity in the second session was significantly greater than in the first. Figure 13 shows the mean values for all subjects, however most individual values for hamstring %MVC ranged from 5-25%. There were two subjects in particular whose large %MVC values in the second session, i.e. greater than 80%, influenced the hamstring mean and standard deviation values.

CHAPTER 5

DISCUSSION

The results of the current study demonstrate that the bent knee AIS technique increases knee extension ROM. The right leg ROM increased within the first session and between the first and second sessions. Our results are similar to those found in previous AIS investigations. Both Leimohn et al. (1999) and Middag and Harmer (2002) found significant increases in ROM following short term AIS training programs of three weeks. A 13-week study by Marino et al. (2001) found a significant improvement in the goniometric measurements of ROM after both 7 weeks, and 13 weeks, although the general assessment of flexibility, a sit-and-reach test, remained unchanged.

In the present study stiffness values remained unchanged in spite of the AIS intervention. These results are similar to those obtained by Halbertsma and Goeken (1994) and Halbertsma et al. (1996) who studied CR and static stretch techniques. These studies suggested that the observed increase in hamstring extensibility (ROM) could only be achieved by either a change in the elasticity (stiffness) of the muscle or by an increase in pain tolerance. In 1994 Halbertsma and Goeken could not establish a significant change in the stiffness of the hamstrings following a 4-week PNF CR stretch program. In 1996 Halbertsma et al. found that there was no significant change in the course of the passive muscle stiffness curve following a 10-minute static stretch, with respect to the pre-stretch stiffness curve. Therefore, both the investigations concluded that the increase in

ROM was caused by an increase in stretch tolerance of the subjects. Neither of these studies considered the effects of neural components, another identified mechanism which may explain the increases in ROM (Etnyre & Abraham, 1986b).

An investigation into static stretching by Magnusson et al. (1996a) found that repeated stretches resulted in decreased stiffness, although the effects were transient as the stiffness values returned to baseline within one hour. They concluded that biomechanical variables may be acutely altered by stretch training, however the long-term effects of stretching remain unclear. Further investigations by Magnusson et al. (1996b; 1996c) also found that both short-term and long-term stretching had no effect on stiffness values. They concur with the studies by Halbertsma et al. (1994; 1996) reporting that the increase in ROM achieved from stretching is an increase in stretch tolerance rather than a change in the mechanical or viscoelastic properties of muscle. The mechanism for an altered stretch perception following stretching is unknown. It has been reported that pain perception can be affected during dynamic exercise, so it is possible that a similar response may be stimulated by active stretching. Another possibility is that nociceptive nerve endings in the joint and muscle play a role via neurotransmitter modulation or gate control (Magnusson, 1996b).

Because EMG was collected during the performance of AIS in the lab, it is possible to consider the potential contribution of neural mechanisms to our findings. Our data show that the hamstring muscles were significantly less active than the VL muscle during AIS, and that reciprocal inhibition may be occurring. In

the bent-knee hamstring stretch the VL muscle actively contracts to extend the knee which stimulates the muscle spindles of the VL. A synapse in the spinal cord with a sensory neuron sends an inhibitory signal to the antagonistic muscles, the hamstrings. Thus, the hamstrings are in a relaxed state, optimizing their ability to lengthen. Investigations of reciprocal inhibition have found mixed results. Some research (Moore & Hutton, 1980; Condon & Hutton, 1987; Osternig et al., 1987; Osternig et al., 1990) has found that an increase in ROM is independent of muscle activity. They reported that stretch procedures that produced the greatest increase in ROM, such as CRAC, were accompanied by relatively high levels of activity in the muscles being stretched. Therefore, they concluded that full muscle relaxation was not imperative for effective stretching. The results of our study are in agreement with research that indicates that reciprocal inhibition may be a contributing factor to the effectiveness of a stretch technique (Shindo et al., 1984; Etnyre & Abraham, 1986b; Etnyre & Abraham, 1988; Guissard et al., 1988; Guissard et al., 2001). A potential reason for the disagreement in the literature may be due to the instrumentation and method of analysis used in the investigations of the neural mechanisms. The studies which have shown reciprocal inhibition to be a contributing factor have monitored the H-reflex of the muscles. The H-reflex is an electrical stimulation of a nerve that recreates the myotatic stretch reflex that occurs when a muscle is stretched (Palmieri, Ingersoll & Hoffman, 2004). The H-reflex is a specific tool to assess neurological function. The studies which have disregarded the significance of reciprocal inhibition have only used surface EMG. Surface electrodes are very

effective for studies of general muscle activity, for example the progression from relaxation to tension in a muscle contraction. However, they are only effective for studying superficial muscles and cannot detect signals from small muscles (Basmajian & Deluca, 1985). An investigation by Etnyre and Abraham (1988) assessed muscle activity of the agonist and antagonist muscles during PNF stretch procedures using both fine wire and surface electrodes. Examination of recordings from the wire electrodes showed no activity in the antagonist muscle during agonist contraction. This indicated that reciprocal inhibition occurred during the stretch technique. EMG recorded with surface electrodes contained inter-muscle cross-talk and therefore appeared to show suppressed reciprocal inhibition phenomena. The researchers suggested that care must be taken when making conclusions about muscle activity, if surface electrodes have been used (Etnyre & Abraham, 1988). Our study used surface electrodes to assess muscle activity but found results which support research which used in-depth, specific methodology to test neural function. We conclude that neural mechanisms, such as reciprocal inhibition, possibly played a role in AIS as assessed in this study. However, further research, specifically assessing the H-reflex, is required to confirm the neural implications.

The increase in ROM of our control leg was unexpected. However, there are studies which support this finding and offer an explanation for why it occurred. Stretch studies are designed to compare values between a control and a stretch condition. The control can be a separate group of subjects or the contralateral extremity in the same subject. For both methodological designs

there are investigations which report an increase in ROM of the control group or leg. An investigation of static and ballistic stretch techniques found that the control group also showed a significant increase in dorsiflexion ROM (Mahieu et al., 2007). It was concluded that the increase in ROM of the control was due to a learning effect. They believed that the subjects were able to achieve a greater ROM in the second session as a result of the practice they had received in the initial test session. Additionally, familiarity with the testing procedures, i.e. a learning effect, may have altered the subjects' stretch tolerance thus allowing a greater ROM for the same perceived stretch sensation. The method of examining the differences between a stretch-trained and a control leg within the same subject population was used by Handel, Horstmann, Dickhuth and Gulch (1997). The measurements of the stretched leg were normalized with respect to the contralateral control leg and then related to the results of the pre-training data. This methodology was used because it was then possible to filter out influences that uniformly affected the state of both legs, such as any general improvements in fitness in the course of the training program or adaptations to the testing procedures. We performed an ANCOVA on our data in order to eliminate any bilateral learning effects or physical changes resulting from the recreational activities that our subjects were allowed to maintain participation in during the intervention program. The between session ANCOVA showed a significant increase in ROM. The 2-way ANCOVA indicated an increase in ROM both within the first session and between the two sessions was close to being statistically significant ($p = 0.063$ and $p = 0.052$ respectively). Our results indicate that long

term AIS is effective at increasing ROM, and a trend for the immediate benefits of AIS is evident.

Alternatively, Grady and Saxena's study (1991) of static stretching observed changes in control leg ROM which were credited to the same mechanisms of action as PNF exercise. The three experimental groups performed 0.5, 2.0 and 5.0 minutes, respectively, of static stretching on one ankle while the opposite ankle served as the control. The increased dorsiflexion seen in the control leg was explained in terms of muscle facilitation and inhibition. Before PNF was used as a stretching technique to increase ROM it was a treatment for restoring strength in patients with neuromuscular disorders. Patients who were unable to exercise an injured limb performed PNF techniques on the unaffected side. The injured limb showed benefits from the unilateral procedures, such as decreased atrophy and a decreased loss of flexibility and strength, indicating a cross over effect had occurred. We speculate that performing AIS on one leg, which follows the same concepts of muscle facilitation and inhibition as PNF, may have a cross over effect. This may be a reason for the increased ROM of the control leg.

Chapter 6

CONCLUSIONS

The purpose of the study was to investigate tissue properties of human skeletal muscle in response to the AIS technique for the hamstring muscle group. The study assessed the acute and long term effectiveness of AIS and examined one possible underlying mechanism of action. The AIS protocol used in the study produced statistically significant increases in ROM after a 6-week stretching program. It also produced increases in ROM after a single bout, however it was impossible to determine conclusively whether these changes were statistically significant, due to changes in the control leg ROM. Investigations of other stretch techniques have identified three proposed mechanisms which may explain the increased ROM: mechanical, neural and a change in stretch tolerance. The mechanism of action of AIS does not appear to be by way of mechanical mechanisms because there was no change in the stiffness of the muscle. The contribution of neural mechanisms is evident, and this requires further investigation. Also, an altered stretch perception remains a possibility.

6.1 Limitations of the Study

We used the Biodex isokinetic dynamometer for testing to ensure that the subject was stabilized during test sessions, and could be accurately repositioned for the second session. When assessing the hamstring muscles it is important to stabilize the pelvis during knee extension as the hamstrings are two joint muscles, crossing both the hip and the knee. A change in pelvic position can affect knee extension ROM as it will shorten or lengthen the hamstring muscles (Nuyens et al., 2000). However, the design of the dynamometer limited our study to subjects with stiff hamstrings, as flexible persons would reach the maximum knee extension position before feeling a stretch sensation in the hamstrings. It has been noted that subjects who classify as having tight hamstrings on a toe-touch test have both stiffer hamstrings and a lower stretch tolerance than those who classify as having normal hamstrings (Magnusson et al., 1997). Therefore, the results of our study may be limited to persons with tight hamstrings. In addition to limiting the subjects whom we could test, the design of the dynamometer impacted the effectiveness of the AIS procedure during the second test session. We found that subjects' knee extension ROM improved over the 6-week training program. As a result, at the second test session during the AIS procedure some subjects were able to extend their knee to the maximum extension position without feeling the required stretch sensation of 8 out of 10. They reported that they could feel a slight stretch but it was not as strong as what they felt in the first test session. This confirmed that the 6-week AIS program had affected the subjects' knee extension abilities but also may have limited the acute

effect of the technique in the second session. The decreased stretch stimulation may have contributed to the lack of change we found in the stiffness values during this test session. Subjects attained a greater knee extension position while performing the stretch procedure compared to when they simply extended their knee, therefore the passive resisted torque and ROM values were unaffected by this problem.

A substance in which the past history of movement characterizes the stiffness or viscosity is said to be thixotropic. This term is commonly used to describe gels which become fluids when shaken or stirred but which regain their original high viscosity after they are left to settle (Hagbarth, Hagglund, Nordin, & Wallin, 1985). Lakie, Walsh and Wright (1984) reported that human musculotendinous structures possess thixotropic properties. Thus, a warm up was performed prior to stretching to maximize the ability of the musculotendinous until to lengthen. We ensured that all subjects completed a 5-minute warm up, at a moderate resistance and speed, before testing. However, extensibility can also be affected by the time of day (Halbertsma & Goeken, 1994). We did not test the subject at the same time of day for both sessions.

Because we had anticipated that the control leg ROM would be unchanged throughout our study it was assessed only once each session, after the AIS bout. The unexpected increase in left leg ROM after the 6 week training protocol suggests that control leg ROM should have been measured both before and after the AIS bout in each session, ideally before the right leg ROM was assessed. This would have allowed us to test the covariance effect of the left leg

on right leg ROM at each time point in the experiment. While we can confidently report the covariate effect on the right leg ROM over the 6 week training protocol, we cannot be sure of its effect within sessions.

6.2 Future Investigations

The main focus of our investigation was to assess the mechanical mechanisms associated with AIS. We also assessed neural activity with surface electrodes on the VL and hamstring muscles. Our results indicated that neural mechanisms were present during the stretch procedure. A more specific neural assessment technique, such as the H-reflex, could be used to test the effect of AIS. A more detailed investigation of the neural mechanisms could also be achieved if the time point of application of the overpressure could be identified in the EMG data. Also, by using an electrogoniometer, the amount of agonist and antagonist activity could be analyzed at specific joint positions during the AIS procedure. The large acute change in ROM seen in the first test session could be investigated further by assessing ROM at several time points in the 24 hours following, for example one hour post and 24 hours post stretching.

Most studies vary slightly in their measurement technique and prescription of stretch stimulus volume. It would be beneficial to use our methodology to investigate other stretch techniques, such as static, PNF and dynamic. Then we could directly compare the results of the AIS procedure to those more commonly known procedures.

As previously noted, our test procedures were limited to only subjects with tight hamstrings. A measurement technique that could assess both normal and tight subjects would also be an area of interest for future study.

REFERENCES

Åagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, S.P., Bojsen-Moller, F.& Dyhre-Poulsen, P. (2000). Antagonist muscle coactivation during isokinetic knee extension. *Scandinavian Journal of Medicine and Science in Sports*, 10, 58-67.

Basmajian, J.V. & Deluca, C.J. (1985). *Muscles alive, their functions revealed by electromyography* (5th ed.). Baltimore, Md: Williams & Wilkins.

Condon, S.M. & Hutton, R.S. (1987). Soleus muscle electromyography activity and ankle dorsiflexion range of motion during four stretching procedures. *Physical Therapy*, 67(1), 24-30.

Conroy, B. & Earle, R.W. (2000). Bone, muscle, and connective tissue adaptations to physical activity. In T.R. Baelche, & R.W. Earle (Eds.), *Essentials of strength training and conditioning* (pp.57-72). Champaign,IL: Human Kinetics.

Cornelius, W. L. & Hinson, M.M. (1980). The relationship between isometric contractions of hip extensors and subsequent flexibility in males. *Journal of Sports Medicine and Physical Fitness*, 20, 75-80.

Etnyre, B.R. & Abraham, L.D. (1986a). Gains in range of motion of ankle dorsiflexion using three popular stretching techniques. *American Journal of Physical Medicine*, 65(4), 189-196.

Etnyre, B.R. & Abraham, L.D. (1986b). H-reflex changes during static stretching and two variations of proprioceptive neuromuscular facilitation techniques. *Electroencephalography and clinical Neurophysiology*, 63, 174-179.

Etnyre, B.R. & Abraham, L.D. (1988). Antagonist muscle activity during stretching: a paradox reassessed. *Medicine and Science in Sports and Exercise*, 20(3), 285-289.

Etnyre, B.R. & Lee, E.J. (1987). Comments on Proprioceptive Neuromuscular Facilitation Stretching Techniques. *Research Quarterly for Exercise and Sport*, 58(2), 184-188.

Fillyaw, M., Bevins, T. & Fernandez, L. (1986) Importance of correcting isokinetic peak torque for the effect of gravity when calculating knee flexor to extensor muscle ratios. *Physical Therapy*, 66, 23-31.

Gajdosik, R. L. (1995). Flexibility or muscle length? [letter;comment]. *Physical Therapy*, 75, 238-339.

Gajdosik, R.L. & Bohannon, R.W. (1987). Clinical measurement of range of motion. Review of goniometry emphasizing reliability and validity. *Physical Therapy*, 67, 1867-72.

Grady, J.F. & Saxena, A. (1991). Effects of stretching the gastrocnemius muscle. *The Journal of Foot Surgery,* 30(5), 465-469.

Guissard, N. & Duchateau, J. (2006). Neural aspects of muscle stretching. *Exercise and Sport Science Reviews*, 34(4), 154-158.

Guissard, N., Duchateau, J. & Hainaut, K. (1988). Muscle stretching and motoneuron excitability. *European Journal of Applied Physiology and Occupational Physiology*, 58, 47-52.

Guissard, N., Duchateau, J. & Hainaut, K. (2001) Mechanisms of decreased motoneurone excitation during passive muscle stretching. *Experimental Brain Research*, 137(2), 163-169.

Hagbarth, K.-E., Hagglund, J.V., Nordin, M. & Wallin, E.U. (1985). Thixotropic behaviour of human finger flexor muscles with accompanying changes in spindle and reflex responses to stretch. *Journal of Physiology*, 368, 323-342.

Halbertsma, J.P. & Goeken, L.N.H. (1994). Stretching exercises: effect on passive extensibility and stiffness in short hamstrings of healthy subjects. *Archives of Physical Medicine and Rehabilitation*, 75, 976-981.

Halbertsma, J.P., van Bolhuis, A.I. & Goeken, L.N.H. (1996). Sport stretching: effect on passive muscle stiffness of short hamstrings. *Archives of Physical Medicine and Rehabilition*, 77, 688-692.

Handel, M., Horstmann, T., Dickhuth, H.H. & Gulch, R.W. (1997). Effects of contract-relax stretching training on muscle performance in athletes. *European Journal of Applied Physiology*, 76, 400-408.

Harris, R.T. & Dudley, G. (2000). Neuromuscular anatomy and adaptations to conditioning. In T.R. Baelche, & R.W. Earle (Eds.), *Essentials of strength training and conditioning* (pp.15-24). Champaign,IL: Human Kinetics.

Hartley, A. (2000). *Practical joint assessment: lower quadrant: a sports medicine manual.* Etobicoke, ON: Author.

Herbert, R. & Balnave, R. (1993). The effect of position of immobilization on resting length, resting stiffness, and weight of the soleus muscle of the rabbit. *Journal of Orthopedic Research*, 11, 358-366.

Holcomb, W.R. (2000). Stretching and warm-up. In T.R. Baelche, & R.W. Earle (Eds.), *Essentials of strength training and conditioning* (pp.321-342). Champaign,IL: Human Kinetics.

Holt, L.E., Travis, T.M. & Okita, T. (1970). Comparative study of three stretching techniques. *Perceptual and Motor Skills*, 31, 611-616.

Houglum, P. (2001). *Therapeutic exercise for athletic injuries*. Champaign, IL: Human Kinetics.

Hunter, G.R. (2000). Muscle physiology. In T.R. Baelche, & R.W. Earle (Eds.), *Essentials of strength training and conditioning* (pp.15-24). Champaign,IL: Human Kinetics.

Johns, R.J. & Wright, V. (1962). Relative importance of various tissues in joint stiffness. *Journal of Applied Physiology*, 17(5), 824-828.

Kellis, E. & Baltzopoulos, V. (1996). Gravitational moment correction in isokinetic dynamometry using anthropometric data. *Medicine and Science in Sports and Exercise*, 28(7), 900-907.

Kisner, C & Colby, L.A. (1996) *Theurapeutic Exercise Foundations and Techniques*, 3rd ed. Philadelphia, PA; F.A. Davis Company, p. 151.

Kubo, K., Kanehisa, H. & Fukunaga, T. (2001). Is passive stiffness in human muscles related to the elasticity of tendon structures? *European Journal of Applied Physiology*, 85, 226-232.

Kubo, K. Kanehisa, H & Fukunaga, T. (2002). Effect of stretching training on the viscoelastic properties of human tendon structures in vivo. *Journal of Applied Physiology*, 92, 595-601.

Krabak, B.J., Laskowski, E.R., Smith, J., Stuart, M.J. & Wong, G.Y. (2001). Neurophysiologic influences on hamstring flexibility: a pilot study. *Clinical Journal of Sport Medicine*, 11(4), 241-246.

LaBan, M.M. (1962). Collagen Tissue: Implications of its response to stress in vitro. *Archives of Physical Medicine and Rehabilitation*, 43, 461-466.

Lakie, M., Walsh, E.G. & Wright, G.W. (1984). Resonance at the wrist demonstrated by the use of a torque motor: an instrumental analysis of muscle tone in man. *Journal of Physiology*, 353, 265-285.

Liemohn, W., Mazis, N. & Zhang, S. (1999). Effect of active isolated and static stretch training on active straight let raise performance. *Medicine and Science in Sports and Exercise*, 31(5) Supplement:S116.

Magnusson, S.P. (1998). Passive properties of human skeletal muscle during stretch maneuvers. *Scandinavian Journal of Medicine and Science in Sports*, 8, 65-77.

Magnusson, S.P., Aagaard, P., Simonsen, E.B. & Bojsen-Moller, F. (2000). Passive tensile stress and energy of the human hamstring muscles in vivo. *Scandinavian Journal of Medicine and Science in Sports*, 10, 351-359.

Magnusson, S.P., Hansen, P. & Kjaer, M. (2003). Tendon properties in relation to muscular activity and physical training. *Scandinavian Journal of Medicine and Science in Sports*, 13, 211-223.

Magnusson, S.P., Simonsen, E.B., Aagaard, P., Boesen, J., Johannsen, F. & Kjaer, M. (1997) Determinants of musculoskeletal flexibility: viscoelastic properties, cross-sectional area, EMG and stretch tolerance. *Scandinavian Journal of Medicine and Science in Sports*, 7, 195-202.

Magnusson, S.P., Simonsen, E.B., Aagaard, P., Dyhre-Poulsen, P., McHugh, M.P. & Kjaer, M. (1996a). Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. *Archives of Physical Medicine and Rehabilitation*, 77, 373-378.

Magnusson, S.P., Simonsen, E.B., Aagaard, P., Sorensen, H. & Kjaer, M. (1996b). A mechanism for altered flexibility in human skeletal muscle. *Journal of Physiology*, 497(1), 291-298.

Magnusson, S.P., Simonsen, E.B., Dyhre-Poulsen, P., Aagaard, P., Mohr, T. & Kjaer, M. (1996c). Viscoelastic stress relaxation during static stretch in human skeletal muscle in the absence of EMG activity. *Scandinavian Journal of Medicine and Science in Sports*, 6, 323-238.

Mathieu, N.N., McNair, P., DeMuynck, M., Stevens, V., Blanckaert, I., Smits, N. & Witvrouw, E. (2007). Effects of static and ballistic stretching on the muscle-tendon tissue properties. *Medicine and Science in Sports and Exercise*, 39(3), 494-501.

Marieb, E. (2004) *Human Anatomy & Physiology*, 6th ed. San Francisco, CA: Pearson Benjamin Cummings, p. 283.

Marino, J., Ramsey, J.M., Otto, R.M. & Wygand, J.W. (2001). The effects of active isolated vs static stretching on flexibility. *Medicine and Science in Sports and Exercise*, 33(5) Supplement 1:S10.

Marsden, C.D., Obeso, J.A. & Rothwell, J.C. (1983). The function of the antagonist muscle during fast limb movements in man. *Journal of Physiology*, 335, 1-13.

Mattes, A.L. (2000). *Active Isolated Stretching: The Mattes Method*. Sarasota, FL: Author.

McHugh, M.P., Connolly, D.A.J., Eston, R.G., Kremenic, I.J., Nicholas, S.J. & Gleim, G.W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *The American Journal of Sports Medicine*, 27(5), 594-599.

McHugh, M.P., Kremenic, I.J., Fox, M.B. & Gleim, G.W. (1998). The role of mechanical and neural restraints to joint range of motion during passive stretch. *Medicine and Science in Sports and Exercise*, 30(6), 928-932.

McHugh, M.P., Magnusson, S.P., Gleim, G.W. & Nicholas, J.A. (1992). Viscoelastic stress relaxation in human skeletal muscle. *Medicine and Science in Sports and Exercise*, 24(12), 1375-1382.

Middag, T.R. & Harmer, P. (2002). Active-isolated stretching is not more effective than static stretching for increasing hamstring ROM. *Medicine and Science in Sports and Exercise*, 34(5) Supplement 1:S151.

Moore, M.A. & Hutton, R.S. (1980). Electromyographic investigation of muscle stretching techniques. *Medicine and Science in Sports and Exercise*, 12, 322-329.

National Strength and Conditioning Association. (2000). *Essentials of Strength Training and Conditioning*, 2nd ed. Edited by T.R. Baechle and R.W. Earle. Champaign, IL: Human Kinetics.

Nordez, A., Cornu, C. & McNair, P. (2006). Acute effects of static stretching on passive stiffness of the hamstring muscles calculated using different mathematical models. *Clinical Biomechanics*, 21(7), 755-760.

Nuyens, G., DeWeerdt, W., Spaepen, A., Janssens, L., Ketelaer, P. & Bogaerts, K. (2000). Reliability of torque measurements during passive isokinetic knee movements in healthy subjects. *Scandinavian Journal of Rehabilitation Medicine*, 32, 61-65.

Osternig, L.R., Robertson, R., Troxel, R. & Hansen, P. (1987). Muscle activation during proprioceptive neuromuscular facilitation (PNF) stretching techniques. *American Journal of Physical Medicine*, 66, 298-307.

Osternig, L.R., Robertson, R.N, Troxel, R. & Hansen, P. (1990). Differential responses to proprioceptive neuromuscular facilitation (PNF) stretch techniques. *Medicine and Science in Sports and Exercise*, 22(1), 106-111.

Palmieri, R.M., Ingersoll, C.D. & Hoffman, M.A. (2004). The hoffmann reflex: methodologic considerations and applications for use in sports medicine and athletic training research. *Journal of Athletic Training*, 39(3), 268-277.

Purslow, P.P. (1989). Strain-induced reorientation of an intramuscular connective tissue network: implications for passive muscle elasticity. *Journal of Biomechanics*, 22, 21-31.

Reid, D.A. & McNair, P.J. (2004). Passive force, angle, and stiffness changes after stretching of hamstring muscles. *Medicine and Science in Sports and Exercise*, 36(11), 1944-1948.

Sapega, A.A., Quendenfield, T.C., Moyer, R.A. & Butler, R.A. (1981). Biophysical factors in range-of-motion exercise. *The Physician and Sportsmedicine*, 9, 57-64.

Shindo, M., Harayama, H., Kondo, K., Yanagisawa, N. & Tanaka, R. (1984). Changes in reciprocal Ia inhibition during voluntary contraction in man. *Experimental Brain Research*, 53, 400-408.

Shrier, I. (2004). Does stretching improve performance? a systematic and critical review of the literature. *Clinical Journal of Sport Medicine*, 14(5), 267-273.

Taylor, D.C., Dalton, J.D., Seaber, A.V. & Garrett, W.E.J. (1990). Viscoelastic properties of muscle-tendon units: the biomechanical effects of stretching. *The American Journal of Sports Medicine*, 18, 300-309.

Wierzbicka, M.M., Wiegner, A.W. & Shahani, B.T. (1986) The role of agonist and antagonist in fast arm movements in man. *Experimental Brain Research*, 63, 331-340.

Williams, P. & Goldspink, G. (1973). The effect of immobilization on the longitudinal growth of striated muscle fibres. *Journal of Anatomy*, 116, 45-55.